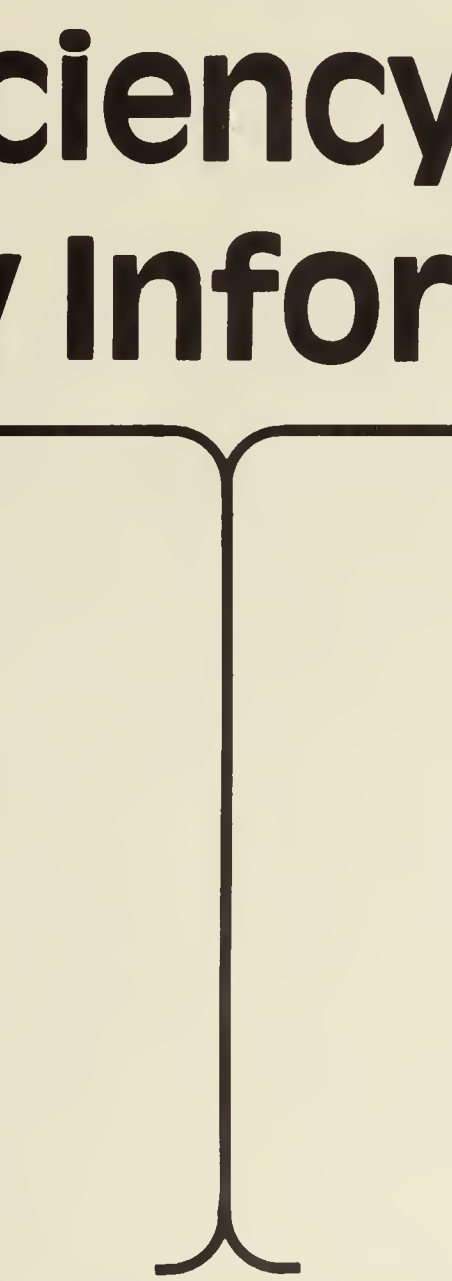
PUBLICATION 5139

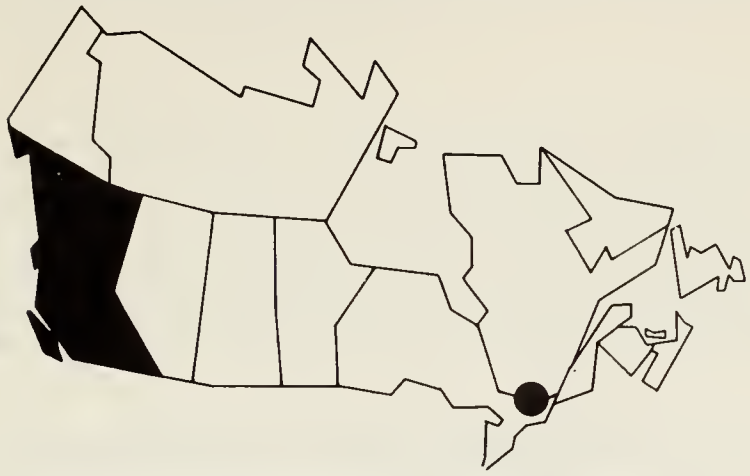
Veterinary Trace Mineral Deficiency and Toxicity Information



Agriculture Canada

Province of British Columbia
Ministry of Agriculture

A FEDERAL/PROVINCIAL PUBLICATION



CANADA / BRITISH COLUMBIA

VETERINARY TRACE MINERAL DEFICIENCY AND TOXICITY INFORMATION

This publication was prepared by the author for the Veterinary Service. Under the provisions of the Federal-Provincial Regional Cooperative Publishing Program, the Canada Department of Agriculture has agreed to print this publication.

R. Puls, Provincial Veterinary Diagnostic Laboratory, Abbotsford, B.C. V2S 4N8

PUBLICATION 5139, available from
Information Services, Agriculture Canada, Ottawa K1A 0C7

Cat. No. A63-5139/1981E ISBN: 0-662-11367-5
Printed 1981 3M—3:81

Aussi disponible en français

F O R E W O R D

This material is designed to aid practising veterinarians, veterinary pathologists and other agricultural advisory personnel in interpreting analytical results received from veterinary analytical laboratories.

The material is not complete and is being continually expanded and updated as time and information become available. The information has been compiled from many thousands of references, some quite conflicting, others complementary.

I have attempted to restrict the information to one page per element per species with emphasis on diagnostic tissue levels. The remainder of the page is devoted to miscellaneous toxicity, treatment or diagnostic data gleaned from the literature. Few references have been specifically cited in the text due to space restrictions, but bibliographies of the references reviewed during the compilation of each section are available from the author.

For further in-depth information, particularly on mechanisms of action, clinical signs, treatment and prevention, the reader is referred to standard texts, some examples of which are listed herein under "General Reference Texts".

R. Puls, N.D.A., C.D.A., P.Ag.
Veterinary Analytical Toxicologist.

TABLE OF CONTENTS

USER NOTES

1. The author considers speed of analysis to be paramount for diagnostic purposes, with ultimate precision of secondary importance.

2. In dealing with diagnostic ranges, which are not absolutely clear cut, wet weight analyses are considered to be sufficiently accurate.

3. Tissue levels should not generally be used as the sole diagnostic criteria unless they fall well within a clearly defined range. (Severe liver damage such as cirrhosis can lead to mineral levels not reflective of dietary intake).

4. Levels falling at the extremes of ranges or in overlapping ranges should be supplemented with additional confirmatory data prior to establishing a firm diagnosis (exposure, clinical, gross and histopathological signs).

5. More than one diagnosis may be warranted for a case (e.g. bacterial infection due to reduced immune response resulting from selenium deficiency).

6. More than one mineral may be involved in a case - in B.C. copper and selenium deficiency often occur concurrently.

7. Interactions should not be overlooked (e.g. molybdenum induced copper deficiency or lead and cadmium induced selenium deficiency.)

8. Repetition within the notes has been avoided whenever possible - the notes listed under all species should be read regardless of the particular species currently under scrutiny.

9. Some of the notes are taken from single unsubstantiated research reports. They are subject to continual revision and updating as more data becomes available.

10. Interactions have not been presented in this edition with any reference to their severity or significance. Some interactions (copper-molybdenum) are severe, others (copper-selenium) are less significant.

INTERPRETATION OF DATA

1. Most tissue levels are presented on a wet weight basis in parts per million (ppm wet wt).

   Wet wt x 3.5-4.0 = approximate dry wt for most tissues.

   Wet wt x 5.0-6.0 = approximate dry wt for fetal tissues.

2. Dietary levels and some tissue levels (hair, bone) are presented on a dry weight basis in parts per million (ppm).

3. All kidney levels refer to cortex.

4. Food consumption (dry matter) for cattle varies from 1.4 - 3.0 percent of body weight. Older fleshier beef cattle consume the lowest amounts. Assuming a dry matter intake of 2.6% of body weight and a body weight of 600 kg (1323 lb)

   100 ppm diet = 2.6 mg/kg body wt = 1.56 g/day.

5. Overlapping ranges indicate that this particular analysis is not a reliable indicator of the status of the animal in the areas of overlap. In general, tissues falling into this category have been omitted from the tables - some commonly analyzed tissues such as liver and kidney have been included.

6. Intermediate ranges:

   (a) Figures falling between the adequate and high ranges indicate levels well in excess of requirement but not approaching a toxic range.

Interpretation of Data contd.

b) Figures falling between marginal and adequate indicate an uncertain area and could fall into either group.

5. Upper ranges for toxicity and lower ranges for deficiency have in many cases been included. The ranges should be considered open ended - the values included are the highest or lowest figures reported in the literature or by personal communication, and have been included to give analysts some idea of the maximum or minimum levels likely to be encountered.

DEFINITION OF TERMS

Deficient: levels at which clinical or pathological signs of deficiency should be apparent.

Marginal: levels at which subclinical effects may prevail, such as reduced immune response, or reduced growth rate.

Adequate: levels sufficient for full functioning of all body mechanisms with a small margin of reserve to counteract commonly encountered antagonistic conditions.

High: levels elevated well above normal but not necessarily toxic.

Toxic: levels at which subclinical, clinical or pathological signs of toxicity would be expected to occur.

Normal: used where deficiencies are unknown, indicates normal background levels.

ABBREVIATIONS

ppm	parts per million
µg/g	micrograms per gram (= ppm)
mg/kg	milligrams per kilogram (= ppm)
ng/g	nanograms per gram (= ppb - parts per billion)
µg/L	micrograms per liter
mg/L	milligrams per liter
µg%	micrograms per 100 ml
mg%	milligrams per 100 ml
g%	grams per 100 ml
mEq/L	milliequivalents per liter
n mol/L	nano moles per liter
µ mole/ml	micro moles per ml
<	less than
>	greater than
LD 50	Minimum dose that will kill 50% of exposed animals
IU/L	International Units per liter
SF/ml	Sigma-Frankel Units per ml
Hb	haemoglobin
BUN	blood urea nitrogen
CPK	Creatine phosphokinase
ɣGT	gamma-glutamyl transferase (transpeptidase)
GSH-Px	glutathione peroxidase
SGOT	serum glutamic oxalacetic transaminase

GENERAL REFERENCE TEXTS

The Merck Veterinary Manual, 5th Edition
Otto H. Siegmund, editor.
Merck & Co. Inc., Rahway, N.J., U.S.A. 1979.

Trace Elements in Human and Animal Nutrition
4th Edition, E.J. Underwood.
Academic Press, New York. 1977.

Veterinary Toxicology, 1st Edition.
E.G.C. Clarke and M.L. Clarke.
Bailliere Tindall, London. 1975.

Clinical and Diagnostic Veterinary Toxicology
Wm.B. Buck, G.D. Osweiler and G.A. van Gelder,
Kendall/Hunt Publishing Co., Iowa. 1973.

Veterinary Medicine, 5th Edition.
D.C. Blood, J.A. Henderson and O.M.Radostits.
Bailliere Tindall, London. 1979.

Trace Element Metabolism in Animals.
C.F. Mills, editor.
E & S Livingstone (Edinburgh & London). 1970.

Trace Element Metabolism in Animals - 2.
W.G. Hoekstra, J.W. Suttie, H.E. Ganther, W.Merts.
University Park Press (Baltimore, London, Tokyo). 1974.

Nutrient Requirements of Domestic Animals series.
National Research Council,
National Academy of Science, Washington, D.C.

FACTORS AFFECTING TRACE MINERAL UPTAKE BY PLANTS

1. Soil pH: lime increases Mo uptake.
   lime decreases Pb, Cd, Cu, Zn, Mn uptake.

2. Plant species: some plants have a greater ability to absorb trace minerals than others. Soil pH has greater effect on some species than others. Clovers by adding N to soils tend to reduce soil pH.

3. Soil organic matter: availability can be affected by reaction with organic matter to form unavailable complexes (Cu, Ni, Fe, Al) to less extent Zn and Mn.
   Conversely some organic complexes are very soluble and probably highly available.

4. Soil type: high clay content reduces Cd uptake.

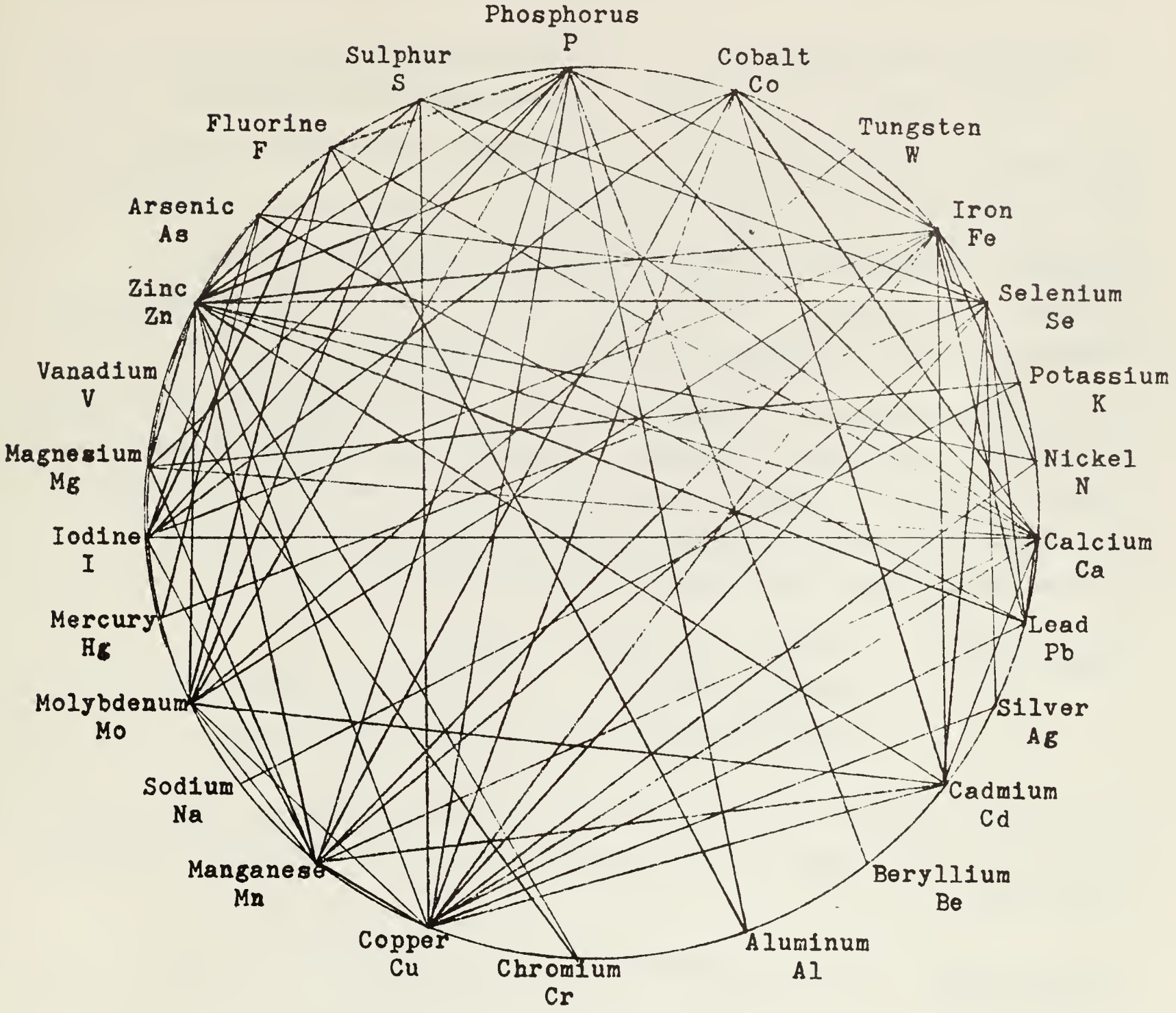
5. Microbial activity: Mn is converted to unavailable form by microbes. Microbial activity reduced below soil pH 5.5 which can release Mn in quantities toxic to plants.

6. Season and climate: trace element absorption varies with stage of growth of plant.

7. Fertilizers: fertilizers can add impurities which increase trace elements supply or reduce their availability (Cd). They can affect the soil pH. They can exert direct effects (P reduces Zn uptake) (P + S increase Mo uptake).

MINERAL INTERRELATIONSHIPS

Modification of diagram by
Jacobson et al, J. Dairy Science, V .55, p935. 1972.



ARSENIC

Cattle: Tissue Levels

Diet	Liver	Kidney	Blood	Urine
Normal	0.03 - 0.40	0.15 - 0.40	0.05	0.5
High	1.0 - 50	1.5 - 5.0		
Toxic - acute	2.0 - 15	3.5 - 38	0.17 - 1.0	2 - 14
- chronic	7.0 - 70	5.0 - 53		
		ppm wet wt		

Normal forage 0.25 ppm — Average soil level 5.0 ppm

Normal hair 0.5 - 3.0 ppm — Normal milk 0.03 - 0.06 ppm

Deficiency - Arsenic may act as an essential trace element but no deficiencies have been identified.

Toxicity - depends on the concentration and the form of arsenic. Trivalent As (arsenite salts) is more toxic than pentavalent As (arsenate salts). Elemental As itself is nontoxic.

Arsenic poinoning is no longer common due to the discontinuation of use of most of the sources of arsenic (insecticides, herbicides, defoliants, sheep dips, etc.). Main sources now are discarded cans of arsenicals and areas of industrial pollution (smelters). Ashes from arsenic treated fence posts that have been burnt contain high level of As.

Toxic dose -		
	Arsenic trioxide	15-45 g
	Sodium arsenite	1 - 4 g (7.5 mg/kg body wt)
	Monosodium acid methanearsonate	10 mg/kg body wt/day

Cattle will develop a tolerance to As if fed at sublethal doses over a period of time. They can subsequently develop a dependence on these high levels.

Interactions - As is antagonistic to I, Se, Hg and Pb.

Signs of poisoning

Sudden death, colic, ataxia, partial paralysis, salivation polydipsia, weakness, watery or bloody diarrhea, depression, trembling, chronic convulsions, hematuria and either hypothermia or fever.

ARSENIC

Dogs: Tissue Levels

	Liver	Kidney	Urine
Normal	< 0.2	< 0.2	0.1 - 0.3
Toxic	> 10.0	> 10.0	> 10

ppm wet wt

Toxicity - Arsenic poisoning in dogs is no longer common due to the discontinued use of arsenical rodenticides.

Hog feed containing a therapeutic level of arsanilic acid may cause chronic arsenic poisoning if fed to dogs.

Toxic dose -

Sodium arsenite - acute single oral dose 50 - 150 mg

Arsenic trioxide - acute single oral dose 100 - 1500 mg

No effect level

Arsenite/arsenate	1.25 mg/kg
Cacodylic acid	30 ppm
Roxarsone (3 nitro)	100 ppm

ARSENIC

Horses: Tissue Levels

	Liver	Kidney
Normal	< 0.4	< 0.4
High	1.0 - 5.0	
Toxic	7.0 - 15	10.0

ppm wet wt

Toxic dose -

Sodium arsonate 1.0 - 3.0 g/day for 14 weeks.
Arsenic trioxide 10 - 45 g single oral dose.

No effect dose -

Arsenic trioxide 0.24 - 0.72 g/day for 2 years

MAXIMUM RECOMMENDED LEVELS OF ARSENIC
IN DRINKING WATER FOR ALL LIVESTOCK
AND WILDLIFE - 1.0 mg/L

For further details on arsenic see:

"Effects of Arsenic in the Canadian Environment", National Research Council Canada - Associate Committee on Scientific Criteria for Environmental Quality, Publication 15391, 1978.

ARSENIC

Pigs: Tissue Levels

	Liver	Kidney	Blood
Normal diet	0.003 - 0.20	0.003 - 0.10	0.01
0.05% cobalt arsanilate	1.0	1.0	
Arsanilic acid (110-250) ppm	2.0 - 4.5	4.0	
Toxic diet	10 - 15	10 - 20	
3-Nitro (toxic)	2.3 - 3.8		

ppm wet wt

Deficiency - arsenic may be an essential trace element - limited evidence to date.

Toxicity - arsenic tolerance in pigs is about 1/10 that of ruminants.

Single toxic dose - Arsenic trioxide 0.5 - 1.0 g
Sodium arsenite 0.05 - 0.15 g

==========

Arsanilic acid

45 - 100 ppm in diet - growth promotion (90 g/ton = 34 ppm As)
200 - 250 - control of swine dysentry
400 - 2000 - toxic after 4 days to several weeks
8000 - toxic in 2 days.

Toxic condition is reversible if feed withdrawn on appearance of toxic signs. Arsanilic acid at acutely toxic levels produces a refusal to eat syndrome.

Signs of Toxicity - inco-ordination, ataxia, apparent blindness, circling. Therapeutic doses of organoarsenical have occasionally proved toxic if the animal is severely dehydrated or debilitated.

==========

3-Nitro-4-hydroxy phenyl arsanilic acid

Recommended dietary level 37.5 g/ton = 6.4 ppm As
Chronic toxic level 190 g/ton or 30 ppm As

Signs of Toxicity - Urination and defecation followed by trembling of the shoulder, ham and back muscles. Later violent tremors, inco-ordination and extreme agitation (screaming with nose resting on ground for support) until animal lies down. At this point trembling ceases, resuming if forced to stand again immediately. When rested for a few minutes animal becomes normal. Signs occur only when stressed.

ARSENIC

Poultry: Tissue Levels

Diet	Liver		Kidney	
	Normal	Toxic	Normal	Toxic
Non Medicated	0.01 - 0.25	5 - 10	0.01 - 0.20	5 - 10
Nitarsone 200 ppm	1.0 - 3.0		0.3 - 2.5	
Roxarsone 45 g/ton	0.7 - 3.5	5 - 10	0.5 - 1.0	3.0
Carbarsone 375 ppm	0.6 - 2.0		0.5 - 1.0	
Arsanilic Acid 100 ppm		5 - 10		5 - 10

ppm wet wt

Nitarsone (4-nitrophenylarsonic acid) 'Histostat'

Maximum recommended level in feed 0.02% (200 ppm).

300 ppm causes chickens to go off feed - some mortality.

600 ppm causes mortality in turkeys.

LD50 chickens 200 mg/kg body wt.

Roxarsone (3-nitro-4 hydroxyphenylarsonic acid)

Maximum recommended level in feed 45 g/ton (14.2 ppm As).

90 ppm depresses growth rate and causes leg weakness.

350 ppm for 3-4 wks. is lethal.

LD50 - 200 mg/kg body wt.

Peak level of arsenic occurs in liver 5-11 days after commencement of treatment (up to 3.5 ppm) levels then drop and plateau at 1.0-1.5 ppm.

Carbarsone (p-ureidobenzenarsonic acid) 'Carbosep'.

Maximum recommend level in feed 375 ppm (0.0375%).

3200 ppm is not toxic to turkeys.

Arsanilic Acid

Maximum recommended level in feed 100 ppm or 90 g/ton (34.3 ppm As).

Turkeys - 400 ppm decrease wt gain, LD50/28 days = 800 ppm

Chickens - 1000 ppm decrease wt gain, LD50/72 days = 1500 - 2000 ppm

ARSENIC

Rabbits: Tissue Levels

Diet	Liver (Total arsenic)
Normal	1.0 - 2.5
Toxic (60 ppm arsanilic acid)	3.0 - 6.0
	ppm wet wt

Toxicity - Arsanilic acid is toxic to rabbits. Younger rabbits are more susceptible than older rabbits.

Chronic toxic dose -

In water: 16 mg/day - adults
9 mg/day - young

8 - 10 mg/kg body wt arsanilic acid

In food - > 60 ppm arsanilic acid

Signs of toxicity -

Weight loss or failure to gain weight.

Profuse watery diarrhea, anorexia and depression.

Occasional nystagmus and epileptiform seizures prior to death.

ARSENIC

Sheep: Tissue Levels

Diet	Liver	Kidney	Blood	Urine
Normal	0.03 - 0.20	0.1 - 0.3		
High	4 - 8		0.04 - 0.08	
Toxic	10 - 50	10 - 40 ppm wet wt	5.0	100 - 150

Sheep and cattle do not find arsenic distasteful and may develop a taste for it. Sheep are slightly more tolerant of arsenic than cattle.

Toxic single dose

Sodium arsenite	11 mg/kg body wt or 0.2 - 0.5 g
Arsenic trioxide	3-10 g
Arsanilic acid	2000 - 4000 ppm in diet.

Arsenic in the most toxic form is tolerated at 10-20 ppm in diet.

Arsenic in the least toxic form is tolerated at 1000 ppm in diet.

Signs of toxicity

Abdominal pain, depression, groaning, salivation, diarrhea.

BROMINE

Cattle: Tissue Levels

	Diet	Liver	Kidney	Serum
Normal	5 - 200	10 - 20	30 - 90	0.60 - 2.0
High	1000 - 2000			10 - 25
Toxic	3000 - 10,000	300 - 1000	300 - 2000	30 - 50
	ppm dry wt			mEq/L

Normal milk (43 ppm diet) 0.13 - 0.25 mEq/L
or 5 - 10 ppm

Toxic signs - lethargy, weakness, ataxia, recumbancy.

Source - hay grown on methyl bromide treated soil.

Horse - as for cattle.
Goat - as for cattle.
Chicks - toxic diet 5,000 - 10,000 ppm (high F intake aggravates Br toxicity.)

Sheep: Tissue Levels

Diet	Liver
8.0	2.0
30	3.4
ppm	ppm wet wt

CADMIUM

Cattle: Tissue Levels

	Diet	Liver	Kidney	Blood
Normal	0.01 - 0.5	0.02 - 1.0	0.05 - 1.5	0.004 - 0.04
High	$\nearrow$ 5.0	1.4 - 2.0	5.0	
Toxic (chronic)	50 - 500	50 - 160	100 - 250	0.04
(acute)	2000 - 3000	50	>200	
	ppm dry wt		ppm wet wt	

	Hair	Muscle	Milk	Bone
Normal	0.04 - 0.60	0.024	0.001 - 0.03	<0.05
High	0.67 - 16.0		0.010 - 0.03	
Toxic	40 - 100			
	ppm dry wt	ppm wet wt	ppm wet wt	ppm wet wt

Cadmium accumulates in tissues as a function of age (liver and kidney). Cadmium does not accumulate in bones or muscle tissue. Hair levels increase in winter, as do levels in grass while it is dormant. Milk levels reported in literature are unreliable due to contamination by milking machines and handling equipment. Cd is not excreted in the milk to any great extent.

Deficiency - There is little evidence to date to indicate Cd acts as an essential trace element.

Toxicity - Dietary levels: 100 ppm cause abortions
200 ppm cause increased BUN in ruminants

Blooc levels are not diagnosticly elevated in toxicity situations. Cadmium will cross the placental barrier to feti only at very high dose levels.

Signs of toxicity - Excess cadmium causes anemia, abortions, still births, malformed feti, impaired growth rate, hypertension, sodium retention and reduces immune response.

Interactions - Cadmium is antagonistic to Cu, Fe, Mn, Se, Co and Zn and vitamin A metabolism. Excess Cd reduces the toxic effect of Pb. Cadmium is more toxic if the level of Ca in the diet is low. Relatively low levels of Cd in the diet of pregnant rats (4.3 µg Cd/ml drinking water) will significantly reduce the storage of Cu and Fe in fetal tissues.

CADMIUM

Dog: Tissue Levels

Diet	Liver	Kidney
Normal 5.0	0.037	0.12 - 0.18
High 30	1.0 - 7.0	4.0 - 17.0
Toxic		> 200
ppm		ppm wet wt

Horse: Tissue Levels

Diet	Liver	Kidney
Normal	0.01 - 5.0	0.05 - 10.0
High	22.0	4.2 - 23.0
Toxic		> 200
		ppm wet wt

Pig: Tissue Levels

Diet	Liver	Kidney
Normal 0.06 - 0.83	0.10 - 0.50	0.15 - 0.99
High 5.0 - 60	3.0 - 30.0	2.0 - 50
Toxic 120		> 270
ppm		ppm wet wt

Rabbit: Tissue Levels

Diet	Liver	Kidney
Normal	0.30	3.6
Toxic		200 - 300
		ppm wet wt

CADMIUM

Poultry: Tissue Levels

	Diet	Liver	Kidney	Egg Yolk	Muscle
Normal	0.01 - 1.0	0.1 - 0.5	0.4 - 1.5	0.02 - 0.10	0.02 - 0.06
High	3 - 20	5 - 10	5.0 - 60	0.06 - 0.10	0.14 - 0.3
Toxic	40 - 1000	15 - 100	70 - 140		0.16 - 0.3
	ppm		ppm wet wt		

Toxicity - LD_{50} wide ranges reported in literature no doubt due to make up of diet.

200 - 500 ppm diet.

165 - 188 mg/kg body weight.

3 ppm in diet has caused nephritis and enteritis yet enhanced egg production. 60 ppm in diet has produced no ill effects. 12 - 60 ppm in diet has reduced feed consumption and egg laying. No effects on fertility of eggs have been identified.

Interactions - Added dietary ascorbic acid protects against Cd induced anaemia. Low Ca and Fe in diet allows increased Cd absorption. Liver and kidney Zn levels increased with added levels of Cd in diet in one trial.

CADMIUM

Sheep: Tissue Levels

	Diet	Liver	Kidney	Wool	Blood
Normal	< 0.5	0.04 - 1.40	0.14 - 0.48	0.55 - 1.22	0.02 - 0.20
High	5.0	2.0 - 20	4.0		
Toxic	50 - 500	50 - 600	50 - 400	> 20	0.10 - 0.20
	ppm	ppm wet wt		ppm dry wt	ppm wet wt

Soil Cadmium -
Normal soil level 0.4 ppm (range 0.1 - 1.0)
Abnormal soil level 160 ppm (proximity of smelter).

Soil contamination - superphosphate fertilizer (the main source of soil contamination) contains an average 42 ppm depending on source of supply.
Municipal sewage sludge may have a high Cd content (range 1 - 3410 ppm Cd).

Soil and Plant Interrelationships
Phosphate fertilizer reduces uptake of Cd by plant.
Plant uptake of Cadmium increases in acid soils.
High clay content reduces uptake.
Uptake varies with plant species.

Soil Cd	Oats	Wheat	Clover
0.03 ppm	0.03	0.01	0.10
0.21	0.27		1.38
	ppm dry wt		

Normal grain	0.01 - 0.07 ppm dry wt
Normal grass	0.8 - 1.7 ppm
High grass	3.6 - 40.0 ppm

CHROMIUM

Rabbit: Tissue Levels

Diet	Brain	Kidney	Liver	Serum
Normal	0.66-0.94	0.42-1.58	0.3-1.0	4.3-5.5
High - Toxic				
-Trivalent Cr	1.0 -1.96	17.0-30.7	6.0-50	9.0-12.0
-Hexavalent Cr	3.81-5.66	3.3-11.2	10 -50	13.0-15.0
	ppm	wet wt		ppm

Level in blood is not a good indicator of body status:
Hair level may be good indicator.

Bovine Milk	8-13 ng/g (average level)
Eggs	0.05 - 0.15 ppm wet wt

Deficiency

Dietary requirements of livestock are unknown.
Chromium deficiency reduces growth and longevity, disturbs glucose, protein and lipid metabolism.
Chromium is thought to function as a cofactor with insulin.

Toxicity

Hexavalent Cr is more toxic than trivalent Cr.
Cats tolerate 1000 mg/day, and rats 100 mg/day trivalent Cr. 5 mg/l Cr III in the drinking water of rats and mice or 20 ppm in the diet produced no ill effects over their lifetime.
Toxic diet approximately 2 mg/kg/day.
BUN levels increase with chronic chromium toxicity indicating renal damage.

Interactions

Cr and Zn are antagonistic.

COBALT

Ruminant: Tissue Levels

	Diet	Liver	Kidney	Milk
Deficient	< 0.06	< 0.005	0.014	
Marginal	0.07 - 0.10	0.005 - 0.017		
Adequate	0.10 - 0.25	0.020 - 0.085	0.071	0.4 - 1.1
High	4 - 20	0.085 - 5.7		
Toxic	> 20	5.0 - 300	30 - 200	
	ppm	ppm wet wt		µg/L

Hair Co is an unreliable indicator of the Co status of animals.

Ruminant: Vitamin B_{12} Levels

	Diet	Liver	Serum
Cobalt	Deficient	0.04 - 0.10	0.04 - 0.20
	Marginal	0.11 - 0.22	0.25 - 0.35
	Adequate	0.25 - 2.24	0.40 - 0.60
		ppm wet wt	ng/ml

Sheep: Blood Levels

	Normal	Cobalt Deficient
Plasma glucose	59 - 72	26 - 36 mg%
Alkaline phosphatase	60 - 90	18 - 44 IU/L
Formiminoglutamic acid	0	0.10 - 0.20 µ mole/ml
SGOT	50 - 70	400 - 600 SF/ml
Blood pyruvate	0.60 - 0.90	1.0 - 2.2 mg%
Ascorbic acid	4 - 8	1.0 - 3.5 mg/L
Pyruvate kinase	40 - 80	200 - 5000 mU/ml

Sheep: Urine Levels

	Cobalt Adequate	Cobalt Deficient
Urinary Methylmalonic Acid	< 25 µg/ml	30 - 150 µg/ml
Urinary Formiminoglutamic Acid	0 - 0.01 µ mole/ml	0.05 - 0.60 µ mole/ml

COBALT
RUMINANTS

Deficiency

Sheep have a higher dietary requirement than cattle - daily requirement 0.08 mg/day for adult sheep.

Signs of Deficiency - ocular discharge, listlessness, anaemia, loss of apetite, loss of condition and weakness. "Ill thrift". White liver disease in sheep appears to be the result of Cobalt or Vitamin B12 deficiency coupled with an unknown entity (plant metabolite or mycotoxin). Cobalt deficiency reduces conception rates in cattle, and in sheep a reduction in estrus occurs.

At the onset of Co deficiency, Vitamin B12 levels fall first (the most sensitive indicator), followed by loss of apetite and elevated pyrurate levels in 10-14 weeks. Other parameter alter after six months on a deficient diet.

Interactions - Cobalt deficiency eventually leads to:
Thiamine deficiency (reduced erythrocyte transketalase levels) reduced plasma ascorbic acid, glucose and alkaline phosphatase levels, elevated pyruvate, pyruvate kinase, SGOT, formimino-glutamic acid serum levels and increased urine methyl malonic acid.

Cobalt deficiency reduces the storage of copper in bovine and possibly ovine livers and occasionally elevates ovine serum copper levels.

Possible Mn, Zn and I antagonism.

Treatment and prevention of deficiency - drench with cobalt chloride or sulphate - effective for about 3 weeks.
Bullet of cobaltic oxide given orally to lodge in reticulo-rumen - effective for at least 3 years.
Treatment of pasture with 1 to 5 kg $CoSO_4$/hectare - effective at least 1 yr.
Free choice mineral or salt licks containing Co (0.004 - 0.01%)
Subcutaneous injection of hydroxocobalamine (2 mg initially then 1 mg/month).

Toxicity - toxicity and deficiency signs are similar - maximum safe daily dose of cobalt chloride for calves 3 - 8 mg. Sheep are less susceptible to toxicity than cattle - toxic diet for sheep is 200 ppm Co.

COBALT

Pigs: Tissue Levels

	Diet	Liver	Kidney	Serum
Deficient				
Marginal				
Adequate	1.0	1.0 - 2.0	0.40	0.17 - 0.60
High	100 - 300	6.0 - 8.0	10 - 17	1.00 - 1.20
Toxic	400	10 - 20	25 - 40	1.20 - 1.70
		ppm dry wt		ppm

Urine: Normal 16 - 20 mg/L High 220 mg/L

Toxic signs - cease eating, gaunt, stiff legged, unco-ordinated, muscle tremors, hump backed. Cobalt and iron are antagonistic. Added iron + manganese + zinc or methionine reduce toxicity.

Toxic diet - 100 mg/kg body wt for 3 days. Maximum safe daily dose of cobalt chloride - 1 mg for weaned pigs.

COBALT

Chickens

Maximum diet 4.0 ppm when less than 10 ppm Fe. 50 ppm is toxic. Additional iron and protein reduces toxicity.

COBALT

Soil - Plant Interrelationships

Liming pasture reduces plant uptake of Co and can induce deficiencies in sheep even when soil levels of Co are 4.8 ppm.

Deficient soil	< 3.0 ppm (< 0.25 ppm acetic acid extractable Co)
Normal soil	> 5.0 ppm

High soil Mn (> 1000 ppm) reduces plant uptake of Co.

COPPER

Cattle: Tissue Levels

Diet	Liver	Kidney	Serum	Hair
Deficient	1.0 - 10.0		0.06 - 0.70	1.0 - 6.7
Marginal	5.0 - 25.0		0.55 - 0.80	6.5 - 8.3
Adequate	25.0 - 150	4.0 - 6.0	0.80 - 1.50	6.7 - 15
High	250		2.50 - 4.0	
Toxic	250 - 700	10.0 - 15.0	4.0 - 11.0	
	ppm wet wt (x 3.5 approx. = dry wt)			ppm dry wt

	Milk	Brain	Serum Ceruloplasmin
Low	0.01 - 0.02 mg/L	4.0 - 9.0	0 - 80
Normal	0.05 - 0.60 mg/L	9 - 18 ppm dry wt	130 - 250 OD units

Liver levels

	Fetus & Newborn	1 month	2 month	3 month
Deficient	2.0 - 30	2.0 - 25	2.0 - 20	2.0 - 25
Normal	35 - 200 (av. 100)	50	35	40

ppm wet wt (x 3.5-4.0 for dry wt)
(fetus x 6.0 for dry wt)

Plasma and whole blood copper levels are similar but plasma or serum levels are better indicators of animals copper status. Not all copper circulating in the blood is available to the animal. Serum copper levels increase from third to fifth month of pregnancy. The author has not found serum levels to be reliable indicators of copper deficiency.

Distribution of copper in the liver is uneven, the caudate lobe having higher concentrations than those found in the dorsal or ventral lobes.

Availability and interactions -

Minimum recommended dietary level copper 10.0 ppm (B.C. hay average 5.0 ppm)

Recommended Cu/Mo ratio 3.0 minimum, 4.3 adequate, 6.0 ideal.

Cattle: Copper, continued

Soil copper levels > 56 ppm - normal pasture.
Soil copper levels < 28 ppm - deficient pasture.
Higher than normal sulphur (from soluble protein), molybdenum, cadmium, zinc or silver content can induce copper deficiency. Molybdenum and total sulphur are synergistic in inducing Cu deficiency.
Cu in silage appears to be less available than that in hay. High nitrate levels in feed may depress copper utilization. Phosphate fertilization of pastures reduces copper uptake by forage plants. Nitrogen and/or Sulphur fertilization of pasture can decrease soil pH, increasing Cu uptake and decreasing Mo uptake. Young grass passes through an animal at a faster rate than old grass; this coupled with higher protein levels reduces availability of Cu in young grass.
At low Cu soil level clinical hypocupraemia was seen only if Mo pasture level was > 5.0 ppm. In hypocupraemic areas with no clinical signs Mo pasture levels were 1-4 ppm.
Copper and other minerals in tall fescue are poorly available to cattle.
Estrogens appear to adversely affect copper absorption or utilization.

Signs and Effects of Copper Deficiency in Cattle - Reduced fertility in cows and semen quality in bulls. Poor hair coat, diarrhea and reduced growth rate. Sudden death. Inability to suckle, incoordination, stiff gait, opisthotonus or lateral recumbancy in newborn and young calves. Cattle can have an extremely high or low copper status without showing any signs of toxicity or deficiency; the reasons for this are not clearly understood.

Entities often occuring concurrently with copper deficiency - either parasitized animals are more susceptible to Cu deficiency or Cu deficient animals are more susceptible to parasitism (probably the latter). Vitamin D deficiency tends to occur in Cu deficient animals with concurrent bone deformities. Magnesium deficiency concurrent with Copper deficiency has occurred frequently in Scotland in single suckled calves. Calcium plasma levels tend to be lower in Cu deficient animals. Copper and Selenium deficiency frequently occur concurrently in B.C. cattle. Copper deficient animals appear to suffer from reduced immune response mechanisms.

Cattle: Copper, continued

Prevention of copper deficiency: Oral

Free choice trace mineral mix or trace mineralized salt (0.1 - 0.2% Cu) mixed into ration where possible to give 10 mg/kg in total feed.

Treatment - Calves: 3fl.oz. 1% $CuSO_4$ solution orally. Oral copper oxide granules (50g) have a longer residual effect (B.D.H).

Parenteral prevention or treatment

Inject 100 mg Cu subcutaneously at 6-7 months pregnancy (lasts 3 months)
100 mg Cu subcutaneously after calving
50 mg Cu subcutaneously to calf at 2-3 months.

Copper Toxicity -

Young calves are more susceptible than older cattle.

Acute toxic dose - calves 40-100 mg/kg body wt
cattle 200-800 mg/kg body wt.

High dietary levels of protein, Zn, Fe and Mo reduce copper toxicity.

COPPER

Dogs: Tissue Levels

	Diet	Liver	Kidney	Serum
Deficient				
Normal	7-10	20.0 - 70.0	3.0 - 20.0	0.60 - 0.80
Toxic		400 - 3000		
	ppm	ppm wet wt		ppm

Serum levels slightly decreased by stress and castration of males.
Serum levels <u>increased</u> by malignancies, chronic infections and epilepsy.
Females have lower serum copper levels than males.
Decreased serum Cu was associated with decreased T. protein levels.

An inherited defect leading to excessive copper accumulation and copper toxicity has been identified in some Bedlington Terriers. (Twedt, D.C. et al, JAVMA, 175(3), 269-275).

<u>Clinical Signs of Copper Toxicosis</u> - acute episodes of anorexia, vomiting, weakness, lethargy and dehydration or indisidious deterioration of general condition followed by jaundice, ascites, cachexia, hepatic encephalopathy and death.

COPPER

Horses: Tissue Levels

	Diet	Liver	Kidney	Serum
Deficient		< 3.5	< 4.0	
Adequate		4.0 - 7.5	7.3 - 9.3	1.0 - 1.70
High	800 ppm	1000-1500	30 - 40	
Toxic				

ppm wet weight basis

Serum copper increases from 30 days before parturition until parturition in mares (up to 2.40 ppm in older - 20 yr mares).

Copper deficiency can cause bone abnormalities in foals.

MILK: Parturition 1.00 ppm - 4 months 0.20 ppm.

Normal fetal liver appears to be in the region of 29 - 110 ppm wet wt.

COPPER

Pigs: Tissue Levels

	Diet	Liver Young-Mature	Kidney	Serum
Deficient	< 5.0	0.80 - 1.02		0.15 - 0.40
Marginal	5.0 - 10.0	4.0 - 7.0	4.0 - 7.0	0.40 - 1.50
Adequate	10 - 20	6.0 - 20.0	7.0 - 10.0	1.70 - 3.0
High	100 - 250	40 - 70	12.0 - 16.0	1.70 - 3.0
Toxic	> 500	150 - 15000	300 - 1200	4.5 - 77
	ppm	ppm wet wt		ppm

Normal copper	Hair	8.0 - 15.0 ppm
	Liver - newborn	50.0 ppm
	Blood - newborn	1.0 - 1.2 ppm
	Milk - colostrum	2.95 ppm
	23 days	0.92 ppm

	Copper adequate	Copper deficient
Plasma Ceruloplasmin Activity (△ OD 540/min)	0.21 - 0.30	0.01 - 0.10

Mature pigs accumulate more tissue copper than growing pigs.

Dietary levels - Recommended diet - up to 125 ppm is beneficial. 250 ppm questionable for growing pigs but beneficial for mature animals as long as Zn and Fe levels are increased as well. 20 ppm appears minimal for optimum health. Increased incidence of stillbirths have been recorded in Cu deficient pigs.

Interactions - Molybdenum and Sulphate have no appreciable effect on copper metabolism in pigs. Sulphide reduces copper uptake and accumulation in liver. 1800 ppm sulphide in drinking water will prevent Cu accumulation in the liver when fed a diet containing 500 ppm Cu. Increased calcium levels in the diet increase copper storage in the liver. Increased copper levels in the diet increase zinc storage in the liver, but decrease iron storage.

Toxicity - Blood serum is not a good indication of Cu toxicity in pigs. Chronic copper toxicosis can result in iron deficiency anaemia in growing pigs.

COPPER

Poultry: Tissue Levels

	Diet	Liver	Blood	Feathers
Chickens:				
Growers	9- 200	3.0- 15	0.08-0.18	10-15
Layers	10- 130	3.0- 6	0.20-0.45	10-15
Toxic	500-2000	35 -450	0.22-0.38	
Ducks:				
Normal	10- 50	10 - 60	0.22-0.45	
High	100- 200	25 -140		
Toxic	>200			
Turkeys:				
Normal	10- 200	5 - 10	0.18-0.28	
Toxic	500-3000			
Geese:				
Normal		3.0-26.0		
Toxic	300- 600	50 -100		
	ppm	ppm wet wt		ppm

Chickens

Average Cu in normal egg = 60 ug

Average Cu liver day old chick = 6-27 ppm wet wt - level decreases over the first 3 days then increases again to normal. Copper concentration in the plasma of poultry correlates directly with the functional state of the ovary.

Dietary Levels - copper requirement 8-10 ppm diet (positive response to 150 ppm)
copper deficient diet 0.7-4.0 ppm
copper toxic diet > 300 ppm reduces growth rate and egg production.
1000 ppm lethal.

Copper deficiency causes lameness and misshapen eggs in chickens.

Copper toxicity results in increased feather loss, reduction in weight or weight gains and reduced feed intake and egg production.

Interactions -additional zinc prevents accumulation of Cu in liver (Mo does not). Increased Cu supplementation increases the accumulation of Mo, Fe, Zn and Mn in liver. Added methionine protects against Cu toxicity in chicks.

Copper - Poultry contd.

Turkeys

Less susceptible to toxicity than chickens and probably have a higher dietary requirement (60 ppm minimum requirement suggested).

500 - 1000 ppm diet may reduce growth rate and feed consumption.

1500 - 2400 ppm diet reduces growth rate.

3240 ppm lethal in 3 days.

Copper deficiency can result in aortic rupture in turkeys.

4-Nitro medication increases copper requirement.

Ducks and Geese

Accumulate more copper in the liver than chickens or turkeys fed same dietary level. Toxic signs have occurred in ducklings fed a diet containing 200 ppm Cu, but only when subjected to stress.

300 ppm in water is lethal to geese.

Swans

Often accumulate high levels of Cu in the liver (150 - 2300 ppm wet wt). The significance of these levels is not clear. In copper poisoned swans the kidney levels are also elevated (50 ppm wet wt) and the liver levels are over 3500 ppm wet wt.

Mute swans seem to accumulate more copper in the liver than whooper swans.

The Fe:Cu ratio in poultry diets should be 5:1

COPPER

Rabbit: Tissue Levels (Adult)

	Diet	Liver	Kidney	Serum
Deficient	< 4.0	< 3.0		
Marginal	10 - 18	4 - 10	2.5 - 3.8	
Adequate	20 - 100	10 - 50		
High	200			
Toxic				
	ppm dry wt	ppm wet wt		

Rabbits are born with a reserve store of Cu which is drawn upon during the suckling period.
Doe diet of 10 ppm gave average newborn liver content of 37.4 ppm dry wt.

Dietary levels - increasing Cu supplementation from 20 to 200 ppm gives increased rate of gain with 18% protein diet. Only 100 ppm addition required for same results with 22% protein diet.

Interactions - dietary ascorbic acid is antagonistic to copper assimilation in the rabbit.

COPPER

Sheep: Tissue Levels

Diet	Liver	Kidney	Serum
Deficient	0.5 - 4.0		0.10 - 0.60
Marginal	5.0 - 20		0.40 - 0.80
Adequate	25 - 100	4.0 - 5.0	0.80 - 2.0
High	100 - 300		1.5 - 5.0
Toxic	250 - 1000	18.0 - 120	3.3 - 20
	ppm wet wt (x 3.5 - dry wt)		

	Brain	Wool	Milk
Deficient	2.4 - 4.5	0.5 - 1.0	
Marginal	3.5 - 7.0	2.0 - 4.0	
Adequate	5.0 - 10.0	6.0 - 10.0	0.2 - 0.4
	ppm dry wt		ppm

Fetal and neonate tissue levels are the same as the ewe on a dry wt basis. Copper does not accumulate in the fetal liver as it does in cattle. Normal fetal liver 16-20 ppm wet wt basis (x 6.0 = dry wt).

Copper levels in the blood plasma of ewes fall during pregnancy and rise at parturition.

The author has not found serum or plasma copper levels to be a consistently reliable indicator of copper deficiency. Serum copper levels increase with added dietary Mo, this copper is non-ceruloplasmin Cu and is unavailable to the animal.

Dietary requirements - recommended dietary level of copper 5.0 - 10.0 ppm. Maximum safe level of dietary molybdenum 8.0 - 10.0 ppm. Maximum safe level of dietary copper in lamb pellets 20 ppm, or 3.0 mg/kg body wt. Toxic single dose of copper for sheep with adequate stores, 20-50 mg/kg body weight.

Copper deficiency effects - reduced immune response, growth rate, wool pigmentation and length, increased parasitism, bone fractures and wool breaks with loss of crimp. Reduced fertility - failure of implantation, embryonic loss and fetal death have been attributed to Cu deficiency (abortions or still born lambs). Breed differences in susceptability to Cu deficiency and toxicity have been identified.

Copper: Sheep continued

Treatment of Deficiency - Lambs - copper calcium edetate injection, 5-15 mg/month (ewes 90 mg Cu methionate every three months).

Prevention of deficiency - free choice trace mineral salt mix (0.03% Cu). Higher levels may be used for short periods if deficiency is KNOWN to be severe (0.15% for 2 months).
Oral (experimental) 3-5 g CuO needles (ewes), 1-2 g (lambs at 4-5 weeks of age).

Copper storage decreases dramatically with increased parasitism. Care should be exercised in basing a diagnosis of Cu deficiency on tissue levels alone as Cu supplementation coupled with improved parasite control could lead to a toxicity situation.

Interactions - Higher than normal levels of calcium, cadmium, cobalt, ferrous sulfide, selenium and molybdenum plus sulphur from protein, reduce availability and storage of copper in sheep.
Copper retention in the liver is increased by high Mn level in diet. In molybdenum - sulphate induced Cu deficiency kidney levels rise (7-10 ppm wet wt), total plasma Cu remains normal but TCA soluble plasma Cu drops. Growth rate is reduced by Mo induced Cu deficiency but remains normal if adequate sulphur is present.

Toxicity - Pastures fertilized with manure from chickens fed high dietary levels of copper have accumulated sufficiently high copper levels to be toxic to sheep. Pastures fertilized with manure from copper supplemented hogs could also be hazardous to sheep. Mineral supplements designed for cattle generally contain sufficient copper to cause chronic toxicity in sheep. Confinement housed sheep seem to have a lower copper requirement and are more susceptible to toxicity.

Toxicity - treatment of copper loaded sheep - Remove source of excess copper if possible. 500 mg ammonium molybdate + 1.0 g sodium sulphate/ ewe/day for 2 weeks in grain or drench. Reduce to 100 mg ammonium molybdate + 1.0 g sodium sulphate/ewe/day for further 2 weeks.

FLUORIDE

Cattle: Tissue Levels

	Diet	Bone (rib)	Urine	Milk
Normal	10 - 20	200 - 1800	1.0 - 5.0	< 0.16
High	50 - 100	2000 - 6000	5.0 - 15	0.50
Toxic	> 100	6000	20	
	ppm	ppm dry wt, fat free	ppm	ppm

Fluoride is transmitted transplacentally to the fetus.

Fluorine accumulates in bone as a function of age.

Dietary Levels

Dairy cattle (young heifers)	Maximum diet	30 ppm
Slaughter cattle	Maximum diet	100 ppm
Sheep (ewes)	Maximum diet	60 ppm
	Maximum water	10 ppm
(finishing lambs)	Maximum diet	100 ppm
Pigs	Maximum diet	150 ppm
Poultry	Maximum diet	300 ppm

Recent field investigations indicate these maximum tolerance levels may be too high.

Interactions - Fluoride interacts with Al, Ca, P and I, and possibly many others.

Signs of Fluorosis

Chalkiness, mottling, staining, hypoplasia, abrasion and excessive wear of permanent incissor teeth (over 2 years of age).

Lameness, stiffness, treading of the feet, anaemia and hypothyroidism.

Delayed estrus and poor breedability. Stunted growth.

Source of F

Industrial contamination of environment (fertilizer plants and aluminum smelters).

Mineral supplements (high F in some rock phosphate).

Contaminated water supply (1.0 ppm beneficial and 30 ppm toxic).

Deficiency - little evidence to indicate natural occurrence in livestock. 1.0 ppm F in forage seems to be nutritionally adequate.

GENERAL REFERENCES

Effects of fluorides in animals: National Academy of Sciences, Washington D.C. 1974

Environmental Fluoride: National Research Council Canada No. 16081, 1977

Trace Elements in Human and Animal Nutrition: E.J. Underwood Academic Press 4th Ed. pp 347 - 374.

IODINE

Cattle: Tissue Levels

	Diet	Serum		Milk
	Dry-Lactating	Protein Bound (PBI)	Total (TI)	
Deficient	< 0.1	3.0 - 4.0	3.0 - 10.0	8 - 20
Adequate	0.8 - 2.0	4.6 - 12.8	15 - 40	25 - 70
Excessive	10 - 50	30 - 100	90 - 300	510 - 1070
Toxic - chronic	100 - 300		200 - 1500	4700 - 6400
- acute	> 400			
	ppm	µg/100 ml		µg/L

TI correlates well with dietary intake (better than PBI). Calves are born with high total and protein bound I levels in serum which drop to adult levels in 30 - 40 days, the major decline being in the first 8 days.

Toxicity - toxic dose less if animals under stress, 300 - 500 mg/day. Ethylene diamine dihydroiodide (EDDI) is toxic to stressed cattle (50% mortality when stressed with bovine respiratory disease complex). 164 mg I/day may cause stress in cattle. Lactating cows will tolerate 2-3 g I as EDDI per day (150 - 200 ppm I in feed or 3-5 mg/kg body wt). Levels above 2 g I/day as EDDI are toxic to feti.

Acute signs - anorexia, excessive salivation, hyperthermia, coughing, nasal and occular discharge and inappetance.

Chronic toxicity - enlarged thyroid, reduced growth rate, food consumption and milk yield, reduced immune response, rough dry hair coat and decreased fertility, difficulty in swallowing, hacking cough and weepy eye condition, dead or weak calves.

Sources - sea weed, excessive feed additives, water.

Deficiency - reproductive failure - stillbirth, abortions, hairless or weak young. Suppressed oestrus with resultant infertility - goitre.

Causes - low dietary intake.

Normal forage I — Clover 160 - 180 ppb (dry wt); Grass 60 - 140 ppb

Range of 60 - 1500 ppb depending on soil levels.

Interactions

Rubidium reduces I uptake, possibly also As and F. There appears to be a Co - I interaction.

Iodine - Cattle contd.

Thiocyanates (metabolised from clover constituents) and perchlorates are goitrogenic as are most Brassica sp. and many other cruciferous species. Soybean is mildly goitrogenic.

Prevention of deficiency - free choice salt licks or trace mineral supplements containing 0.01% I.

Recommended dosage of Iodine for foot rot prevention - 50 mg/kg

IODINE

Dogs: Tissue Levels

	Diet	Total I	Serum T_3	Serum T_4
Deficient			20 - 100	< 0.5 - 3.7
Adequate	1.5 - 2.0 ppm	5 - 20 µg/100 ml	75 - 200 ng/100 ml	1 - 4 µg/100 ml

Classical hypothyroidism rarely occurs in dogs - Goiter.

Signs of Deficiency - possible patches of alopecia on legs, moth eaten looking coats, refractory skin infection, interdigital pyoderma or seborrhea.

Thyrotropin test - T_3 and T_4 tests sometimes give false normal results. Thyrotropin (thyroid - stimulating hormone (TSH)) response test is often preferred.
Baseline T_3/T_4 levels are determined followed by intravenous administration of TSH (5 units). Four hours later T_4 levels are remeasured. In this interval T_4 levels in the normal dog will triple, whilst little or no increase will be seen in the hypothyroid dog.

T_4 Level	T_3 Level	
1 - 4	75 - 200	Normal (euthyroid)
± 0.6	± 25	Hypothyroid. Treat with T_4
± 2.5	± 25	Inactive peripheral conversion Treat with T_3
± 0.6	± 125	Problems - plenty of active metabolite. Treatment with T_4 is often ineffective.
± 1.4	± 95	Both T_3 and T_4 values are borderline. Try treatment with T_4 for 3 months.
ug/dl	ng/dl	

Values from Veterinary Reference Laboratory, Inc. Newsletter, Vol.2 No. 9.

See Walsh & Brown. Vet Med/SAC, 1980 Feb. pp 223-225

IODINE

Horses: Tissue Levels

	Diet	Serum	
		Total	Protein Bound
Deficient			
Adequate	0.1 - 0.2	2.0 - 10.0	1.6 - 2.7
High			
Toxic	8.8	30 - 50	
	ppm	ug/100 ml	

Fetal thyroid weight - Normal 15 g
Abnormal up to 100 g.

Serum Tri-iodothyronine (T_3)	Normal Levels
Foals	0.5 - 3.7 n mol/l
Adults	0.3 - 2.0
Serum Thyroxine (T_4) All ages	5 - 39

T_4 levels ranging from 0 - 80 n mol/l have been detected in horses with normal thyroid function. Consequently T_4 levels cannot be regarded as a reliable indicator of thyroid function in the horse.

Dietary requirement - pregnant mares require 1 - 2 mg I/day.

Toxicity - 35 - 50 mg I/day or 8.8 ppm in feed fed to a pregnant mare will produce goitrous foals with no signs in the mare.

The use of iodized salt blocks, trace mineral supplements and dairy feed (supplemented with I) in combination for pregnant mares can induce iodine toxicity in newborn foals.

IODINE

Pigs: Tissue Levels

	Diet	Serum Total	Thyroid	Milk
Deficient	< 0.15		1200	
Adequate	0.2 - 2.0	9.3 - 20	1500	0.30 - 1.60
High	25 - 800	1000 - 1800		10.0 -
Toxic	800 - 1600	> 2400		
	ppm	µg/100 ml	ppm dry wt	ppm

Fetal tissues contain 6 times maternal levels.

Toxicity - 400 ppm in diet increases thyroid weight.
800 ppm reduces growth rate, feed intake and liver iron content.
Possible reduced conception at dietary levels in excess of 2.0 ppm.

Deficiency - reduces growth rate and bone maturation, hairless, thickened skin, reduced reproductive performance and lactation.
Possible induction of deficiency by high nitrate in feed.

IODINE

Poultry: Tissue Levels

	Diet	Plasma T_4	Plasma T_3	Egg Iodine
Deficient	< 0.1			2.0 - 4.0
Adequate	0.3 - 1.0	1.7 - 1.8	1.1	8.0 - 12.0
High	10 - 40	2.4		
Toxic	300 - 5000			
	ppm	µg%		µg%

Plasma Thyroxine (T_4) levels increase in salt deficient chickens.

Dietary requirement - 5-9 µg/day or 0.3 ppm.

Toxicity - 300 ppm I in diet reduces egg production hatchability and embryo survival.
5000 ppm I delays sexual maturity in cockerels.
Birds return to normal within 7 days of removing high dietary I.
0.5% KI in water is not toxic to chicks.
Maximum safe level dietary I = 40 ppm

5000 ppm I reduces weight gain in broilers.
Organic and inorganic thiocyanate (from rapeseed meal) reduce I content of eggs.

IODINE

Rabbit: Tissue Levels

	Diet	Serum: Total	Protein Bound
Deficient	0.2		
Marginal	0.2 - 0.3		
Adequate	0.5 - 1.3		
Toxic	200 - 1000 ppm		

Iodine passes placental barrier in rat, guinea pig and rabbit, resulting in fetal blood I levels 2 - 2.5 times those of the mother.

Thyroid weight (adults)	Normal	0.23 g
	Goitrous	2.0 g

Toxicity - Excessively high mortality occurs in rabbit litters when doe is fed 250 ppm I in diet.
500 ppm I in diet results in total loss of litter.

IODINE

Sheep: Tissue Levels

	Diet	Serum		Milk	Thyroid
		Total	Protein Bound		
Deficient	< 0.08	2.5		1.2 - 8.0	20 - 400
Adequate	0.1 - 0.4	3.0 - 12.3	3.0 - 4.0	8.0 - 15	750 - 2500
High	1.0 -	800 - 2600	10 - 95		
Toxic		4000 - 30000			
	ppm	µg/100 ml		µg/100 ml	ppm wet wt

Serum Thyroxine (T_4)	Normal Level	6.0 - 6.6 µg%	80 - 200 n mol/l
	Marginal Deficient	4.7 - 515 µg%	40 - 60 n mol/l
	Severe Deficient level		< 2.6 n mol/l

Deficient ewes may show normal T_4 levels. Normal lambs are born with elevated T_4 levels declining to levels equal to their dams by 8 weeks of age. Deficient lambs are born with T_4 levels similar to or less than those of their dams (the dam levels may be normal).

Toxicity - Chronic signs: hyperthermia and inappetance, goiter.

7 mg I/kg body wt/day or 90 mg/day EDDI or 200 mg/day KI

Acute signs - severe coughing, anorexia, hyperthermia, emaciation, sluggish movements, nasal discharge, reduced appetite, increased respiratory and heart rate.

400 mg/day KI

Some evidence to indicate decreased immune response.

Source - high pasture levels, sea weed, use of supplemented dairy feed and excessive amounts of trace mineralized salt additives designed for cattle.

Deficiency - goiter, reduced fertility and reproductive performance, reduced birth weight and growth rate of lambs, reduced wool growth.

Causes - goitrogenic plants, deficient pastures.

Prevention - free choice trace mineral or salt blocks or mixes containing 0.07% - 0.10% I.

Treatment - 2 oral doses, 280 mgKI or 360 KIO_3 at beginning of 4th and 5th month of pregnancy, or intramuscular injection 2 months prior to lambing of 1 ml iodised poppyseed oil prep (40% I).

IRON

Cattle: Tissue Levels

	Diet	Liver	Kidney	Serum
Low	<40		10-20	
Adequate	100 - 500	45-300	30-150	0.5 - 3.0
High	1000 - 2000	700	300	4.0 - 6.0
Toxic	4000			
	ppm	ppm wet wt		ppm

Deficiency - little data available.

Toxicity - no direct toxic effects have been reported.
10 mg Fe/liter of drinking water has been reported to reduce total water intake by cattle resulting in reduced milk production.

Interactions - Co, Cu, Zn, Mn and Se deficiency could be induced by high levels of iron. Iron toxicity is influenced by Cu, P, Mn and Vitamin E.

IRON

Sheep: Tissue Levels

	Diet	Liver	Kidney
Deficient	<40	15 - 25	13 - 25
Adequate	100 - 280	30 - 300	30 - 200
High			
		ppm wet wt	

Deficiency - confinement raised lambs have shown positive responses in growth rate and immune response to 500 mg iron dextran injections administered at 3 days of age.

IRON

Pigs: Tissue Levels

Normal Levels	Liver	Plasma Iron	Total Iron Binding Capacity	Hemoglobin
Adequate (Adult)	100-200	100-150	200-500	10 -14
At Birth	100-200	70-80	215-221	9.0-14
100 mg Fe intramuscular at day 1-3				
Day 10	100-130	100-150	200-600	10 -14
Day 20	60-100	100-150	200-600	10 -13
Day 30	25-40	100-150	200-600	10 -13
Deficient Levels				
Adult	30-50			4.0-8.0
Day 3 to weaning	10-15	15-60	700-900	4.0-8.0
	ppm wet wt	µg/100 ml		g%

Pigs are born with the same hemoglobin and liver levels as the adult. These levels drop to 70% of the initial value by day 3 with resulting anaemia if no supplementation is given.

Dietary requirement (ppm Fe)	Adult	Growers
Minimum	80 - 100	100 - 150
Recommended	100 - 300	150 - 300
Maximum	2500	
Toxic	4000	

Interactions - dietary ascorbic acid improves Fe absorption.
High levels of Zn, Mn, Cu, Cd, P and I reduce Fe availability.
An iron:copper ratio of 10:1 should be maintained in the diet.

Recommended treatment - Intramuscular injection 100-200 mg Fe as iron dextran at day 1 or 2.
A second treatment at day 15-20 may be beneficial if late weaning or if pigs are not consuming creep feed.
Oral administration of iron does not increase body stores of iron therefore must be fed daily (10-40 mg Fe/day as ferrous fumarate).
Ferrous sulphate appears to be the best bioavailable source of Fe for pig diets.

Iron - Pigs contd.

Feeding high levels of iron to the sow does not prevent anaemia in her pigs unless they have access to her feed or feces.
The effect of injecting the sow prior to farrowing with iron preparations is of doubtful value.
Research conducted at the Wayne Research Center of Allied Mills Inc. indicates that pigs from sows fed a specific amino acid-iron proteinate (Wayne Brood N'iron) remain anaemia free.
Feeding program incorporated 250-800 ppm Fe from Wayne Brood N'iron in the sows diet 30 days prepartum to 14 days lactation.
Milk Fe levels increased from a normal of 1.37 ppm to 2.57 ppm.

Toxicity - toxicities may occur in vitamin E/Se deficient pigs given intramuscular injections of iron dextran.

IRON

Poultry: Tissue Levels

	Diet	Liver	Blood Haemoglobin	Egg (Yolk)
Deficient	15		3 - 5	
Marginal	35 - 45	30	5 - 7	
Adequate	80	66 - 200	8 - 9	134 - 151
High				
Toxic	200 - 2000			
	ppm	ppm wet wt	g/100 ml	ppm dry wt

Added copper will protect against Fe toxicity up to 1600 ppm Fe in diet.
Iron:Copper ratio in feeds should be 5:1 for maximum growth rate.
Ferrous sulphate is the best bioavailable source of iron.
Lead toxicity increases Fe storage in the liver of waterfowl.
High copper levels cause increased Fe storage in the liver.

LEAD

Cattle: Tissue Levels

	Diet	Liver	Kidney	Blood
Normal	<1.0	0.1-1.0	0.2-2.0	0.02-0.20
High	5-20	2-10	3-20	0.30-0.40
Toxic	>100	5-300	10-700	0.35-32.0
	ppm	ppm wet wt	ppm (cortex)	ppm

	Milk	Hair	Bone
Normal	0.002-0.013	0.5-5.0	1.0-7.0
High		60-90	
Toxic	0.10 - 0.25	10-100	
	ppm	ppm	ppm dry wt

The proportion of lead in the liver and kidney varies with the acuteness of the toxicity. It is essential to check both liver and kidney levels as one or the other may not contain diagnostic levels of lead.

Deficiency - little evidence to indicate lead has any function as an essential trace element.

Toxicity - Toxic cumulative dose: 5-7 mg Pb/kg body wt/day (300 ppm diet)
Toxic single oral dose: 200-800 mg/kg body wt.
The toxic level of Pb in forage seems to be between 5-300 ppm. Uncertainty probably due to element interactions.

Interactions - Pb affects copper, iron and selenium metabolism. It is known to decrease immune response and vitamin E utilization (possibly by inducing Se deficiency). Increased calcium may reduce Pb toxicity.

Sources of Lead - "Lead free"paint can contain up to 1% Pb. Other sources: waste engine oil, putty, roofing tiles, lead batteries, industrial pollution, automotive exhaust.

Signs of toxicity - anemia, anorexia, fatigue, depression, constipation or diarrhea, abdominal pain, nephropathy, blindness, head pressing, loss of weight, abortion.

LEAD

Dogs: Tissue Levels

Diet	Liver	Kidney	Blood	Hair
Normal	0.1-3.5	0.1-2.5	0.01-0.20	0-88
High	3.6-5.0	5.0-10.0	0.30-0.80	60-87
Toxic	50 -200	10.0-50.0	0.60-7.4	88-
		ppm wet wt		ppm

Urine	Lead	Delta-Aminolevulinic Acid
Normal	0-50	0 -0.0041
Toxic (before therapy)	> 75	0.016-0.020
(24 hrs after chelation therapy)	> 820	0.015-0.040
	µg/L	mg/mOs

Diagnosis of clinical lead poisoning in the dog should be based on a combination of clinical signs and laboratory data as blood lead levels are not always elevated.

Dogs residing in cities or close to heavily travelled highways often have lead levels close to the upper limit of the normal range.

Lead accumulates in the ends of the long bones and in the hair of chronically poisoned dogs.

Toxic dose - Chronic 3 - 30 mg Pb/kg body wt/day
Acute 600 - 1000 mg Pb/kg body wt/day

Young dogs are more susceptible to lead poisoning due to their habit of chewing during teething and bizarre eating habits.

Signs of toxicity - (similar to canine distemper).

Gastrointestinal - vomiting, colic and occasionally diarrhea.
Nervous - hysteria, clonic-tonic seizures, nervousness, head pressing, opisthotonos, champing of the jaws, inco-ordination, apparent blindness, deafness.

The finding of many nucleated erythrocytes in blood without evidence of severe anemia is considered to be nearly pathognomonic of lead poisoning.

LEAD

Horses: Tissue Levels

	Diet	Liver	Kidney	Blood
Normal	1.0	0.5	0.5	0.04-0.25
High	30-50	3.0-5.0	3.0-5.0	0.30-0.60
Toxic - chronic	100-800	4.0-50	5.0-140	0.33-0.50
- acute	1000	10 -500	20 -200	0.60-2.5
	ppm	ppm wet wt		ppm

	Brain	Urine	Milk	Bone
Normal	0.5	0.04-0.20	0.006-0.013	3.0-4.0
High	1.0-5.0			8.0-10.0
Toxic	10 -30	0.50-5.0	0.28 -0.54	40 -200
	ppm	ppm (after chelation therapy)	ppm	ppm dry wt

Blood levels are not good indicators of toxicity - fatalities have occurred at 0.30 ppm, conversely levels of 0.60 ppm have been recorded in animals showing no toxic signs.

Toxic signs - Pharyngeal and laryngeal paralysis (roaring)
Reduced immune response (possibly due to induced selenium deficiency).

Toxic cumulative dose -

2.4 - 7 mg Pb/kg body wt/day from natural source.

Inorganic lead appears to be less toxic than lead occurring in naturally contaminated forage.

Interactions - Cu, Fe and Se metabolism adversely affected by lead. Pb and Zn appear to be synergistic.

LEAD

Poultry: Tissue Levels

Chickens

	Diet	Liver	Kidney	Blood
Normal	1-10	0.1-0.5	0.1-1.0	0.04-0.05
High	200-1000	5.0-10.0	5.0-12.0	2.0 -6.2
Toxic	5000	18 -90	20 -150	4.0 -12.0
	ppm lead (acetate)	ppm wet wt		ppm

	Brain	Bone
Normal	< 1.0	< 50
High	7.0 -10	150-400
Toxic	12.0-15	>400
	ppm	

Chickens are far more resistent to lead poisoning than waterfowl.

Toxic dose - Chronic lethal dose 320 mg/kg body wt/day for 11-20 days.
1000 ppm lead (as acetate) depresses growth rate.

Signs of Toxicity - drowsiness, thirst, weakness, loss of appetite, diarrhea, anemia, anorexia, peripheral paralysis.
Lead at high levels reduces immune response.

Turkeys - no information.
Ducks - see waterfowl.
Quail and Pheasants - similar to chickens.

LEAD

Sheep: Tissue Levels

	Diet	Liver	Kidney	Blood
Normal	< 1.0	0.05-0.80	0.10-0.80	0.02-0.25
High	3-60	5 -25	5 -100	0.70-0.90
Toxic	5-300	10 -100	5 -200	1.0 -5.0
	ppm	ppm wet wt		ppm

	Bone (Tibia)	Brain	Wool
Normal	1.0 - 3.0	0.1 - 0.5	4.0 - 7.0
High	10 - 40	1.2 - 2.0	12.0-18.0
Toxic - chronic			25.0 -
	ppm	ppm wet wt	ppm dry wt

Toxic dietary level -

Maximum no effect level	0.3 mg/kg body wt/day
Minimum toxic level	3.0 mg/kg body wt/day
Maximum tolerated dietary level	3.0 - 10.0 ppm
Minimum single toxic dose	60 - 80 mg/kg body wt

Lead poisoning in sheep is not common.

Clinical effects -ɣ-aminolaevulinic acid-dehydratase levels in blood begin to fall with ingestion of nore than 0.3 mg Pb/kg body wt/day.
Urinary ALA increases after 10 weeks dietary Pb of 3.0 mg/kg body wt/day.

Interactions - Lead reduces copper storage and selenium metabolism.

LEAD

Waterfowl: Tissue Levels

Diet	Liver	Kidney	Blood
Normal	0.05-0.50	0.1-1.0	0.02-0.50
Toxic:			
Ducks & Swans	10 -64	60 -1600	-33
Geese	9 -102	8 -55	3.3 -16
		ppm wet wt	

Diet	Brain	Bone	Feathers
Normal	0.5-1.8	2.0-32	< 1.0
Toxic	3.0-40	20-300	10-100
	ppm wet wt	ppm dry wt fat free	ppm

Toxic dose - 12 mg/kg/day chronically toxic to ducks.
25 ppm in diet maximum no effect level.
8 pellets #6 shot ingested - lethal to ducks (15 days)
25 pellets #6 shot ingested - lethal to geese (15 days)
5 pellets #4 shot ingested - lethal to geese (15 days)

Signs of Poisoning - Flaccid paralysis and prostration, emaciation, impaction of proventriculus, distension of the gall bladder, green-brownish staining of the gizzard lining, green discoloration of the liver and intestinal tract, and a green diarrhea.

Interactions - Lead interferes with Cu, Zn, Fe and Se metabolism. Elevated iron and zinc levels usually occur in Pb poisoned waterfowl. Absorption of lead from the digestive tract is increased by a high fibre diet, presence of Vitamin D_2 and starvation.
The accumulation of lead in the organs increases with a low calcium diet.

MAGNESIUM

Serum Magnesium

	Cattle	Sheep	Goat	Horse
Deficient	0.1-1.8	0.5-1.5		< 2.0
Normal	2.0-3.0	2.0-2.8	2.8-3.6	2.2-2.8

mg%

	Pigs	Cats	Dogs	Rabbits	Poultry
Deficient				< 1.8	
Normal	2.7-4.0	1.8-2.7	1.8-2.4	2.0-2.1	1.8-3.5

mg%

Urine Magnesium

	Cattle
Deficient	20
Inadequate	20 - 100
Adequate	100- 250
Excessive	300- 400

mg/L

Tissue levels do not represent the magnesium status of the animal.

Deficiency - Hypomagnesemia in dead animals cannot be substantiated chemically unless urine can be obtained.

Daily urinary excretion of magnesium is a better criterion of magnesium supply than serum concentration. Serum levels do not fall until the animal is severely deficient. Urine levels fall immediately the diet becomes deficient.

Classical grass tetany does not usually occur in cattle until serum levels drop below 1.1 mg%. Levels below 2.0 mg% are considered hypomagnesemic.

Occurrence - magnesium deficiency generally arises when grass grows rapidly and is rich in nitrogen and potassium. Susceptibility increases with the age of the animal.

Signs and effect - irritability, tetany, convulsions. Non-clinical hypomagnesemia adversely affects milk yield and heart function.

Magnesium contd.

Toxicity - excess dietary magnesium in cattle reduces feed intake, retards growth and produces diarrhea and emaciation. Dietary levels greater than 1% magnesium (as oxide) are toxic. Levels greater than 2% produce bloody diarrhea and at 4% reduce feed consumption to 25% of normal.

MANGANESE

Cattle: Tissue Levels

	Diet	Liver	Kidney	Hair
Deficient	1.0	1.0		1.0-5.0
Marginal	10-20	1.5-3.0	0.93-1.2	5.0-15
Adequate	40-200	2.5-6.0	1.2 -2.0	10 -80
High	1000	120-230		80
Toxic	2000			
	ppm dry wt	ppm wet wt		ppm dry wt

	Blood	Serum	Milk
Deficient			
Marginal	0.020	0.005	
Adequate	0.07-0.09	0.006-0.03	0.020-0.040
High			
Toxic			
		ppm wet wt	

Bovine fetal and calf levels are generally low (<1.0 ppm wet wt) seemingly reaching normal levels by the 5th day of age.
Monitoring feed levels seems to be the best diagnostic aid.
Red and black hair contain more Mn than white - levels increase when cattle are on pasture irrespective of total dietary intake.

Deficiency signs - Mn deficiency linked to silent heat, reduced conception, abortions, reduced birth weight, increased percentage of male offspring, paralysis and skeletal damage.

Toxicity - indicated by reduced growth rate.

Interactions - Iron, cobalt and calcium are antagonistic to Mn in all species.
High Mn intake may cause calcium retention in feti.
High Mn intake increases I excretion and Fe absorption.
There is a Mn-choline interaction that may be of significance in the "fat cow syndrome".

MANGANESE

Pigs: Tissue Levels

	Diet	Liver	Kidney	Blood
Deficient	0.6	0.53-0.97	0.35-0.45	0.006-0.010
Marginal	6.0-20	2.8 -3.1	0.75-1.13	0.010-0.012
Adequate	40 -50	3.0 -4.0	1.5 -2.0	0.040
High	500-1000	4.0 -5.0	2.0 -3.0	
Toxic	4000			

ppm wet wt

Rabbits: Tissue Levels

	Diet	Liver	Kidney	Hair
Deficient	0.63	0.2-0.3	0.3-0.8	0.25-0.75
Marginal				
Adequate	50	1.0-2.0	2.0-3.0	0.40-1.20
High				
Toxic				

ppm wet wt

Tissues of all species remain relatively constant over a wide range of dietary intakes.

Monitoring feed intake seems to be the best means of ascertaining the Mn status of all species.

Interactions - Low manganese intake reduces the accumulation of Se in pig tissues.
High manganese may decrease iron storage in the body.

MANGANESE

Poultry: Tissue Levels

	Diet	Liver	Blood	Feathers	Eggs	Yolk
Deficient	1 -10	1.5-4.0		1.2	0.5	2.0
Marginal	10-20		0.030-0.048			4.0
Adequate	40-100	2.0-4.0	0.085-0.091	11.0		33
High	1000					
Toxic	4000					
	ppm dry wt		ppm wet wt			ppm

Egg yolk levels are 4-5 times those in the egg white.

Deficiency - reduces body weight gain and causes hock disorders.

Interactions - there is a significant interaction between dietary manganese and socium chloride in turkey poults.
Excess dietary copper slightly increases Mn storage in the liver.

MANGANESE

Sheep: Tissue Levels

	Diet	Liver	Kidney	Wool	Blood
Deficient	6.0	1.0-2.1	1.0-1.2	1.5-6.0	0.010-0.020
Marginal	8 -20		0.5-1.2		0.012-0.014
Adequate	40 -100	2.0-4.4	0.8-2.5	8.0-18	0.020-0.025
High	400-500	2.0-3.0	2.0-2.5		0.030-0.040
Toxic	1000	5.0-10.0	5.0		
	ppm dry wt	ppm wet wt			ppm

Lambs are born generally with high liver stores.

Interactions - High Mn intake may cause increased copper retention in sheep and calcium retention in feti.

Plant uptake - Pasture Mn uptake is reduced by liming. Corn silages are generally low in Mn.

MERCURY

Cattle: Tissue Levels

	Diet	Liver	Kidney	Blood
Normal	<0.01-0.10	<0.01-0.06	<0.01-0.09	< 0.10
High	1.0 - 5.0	2.0 - 40	14 - 146	0.2-3.0
Toxic	4.0 - 30	2.0 - 40	50 - 200	3.0-6.0
	ppm	ppm wet wt		ppm

	Brain	Milk	Hair
Normal	< 0.1	3 - 10	0.1
High			1.0-55
Toxic	0.5-20		5.0-50
	ppm wet wt	ppb	ppm dry wt

Hair levels correlate well with tissue levels.
Methyl mercury is more widely distributed in the body than inorganic mercury.

Toxicity - High tissue residues without toxicity signs have been shown to occur experimentally. Therefore a diagnosis of mercury toxicity should not be based solely on tissue residues.

Blood, urine, fecal and milk mercury levels appear to bear little relationship to mercury toxicity until tissues have reached a saturation level and damage is excessive.

Toxic dose - 0.1 mg Hg/kg body wt of methyl Hg is tolerated by calves for 90 days.
0.2-0.4 is tolerated by calves for 75 days.
Methyl mercury is more toxic than inorganic mercury.

Toxic signs - Ataxia, neuromuscular inco-ordination followed by convulsions and a moribund state. Renal failure.
Time from ingestion to death averages 20 days.

MERCURY

Cats: Tissue Levels

	Diet	Liver	Kidney	Blood
Normal	<0.05-0.30	0.01 - 0.10	<0.01 - 0.10	0.01 - 0.30
High	0.5 -0.8	5.0 - 30	5 - 20	0.30 - 5.0
Toxic	>1.0	30 - 100	20 - 30	6.0 - 20
	ppm methyl Hg		ppm wet wt	

	Brain	Fur
Normal	0.01 - 0.10	1 - 8
High		8 - 65
Toxic	10 - 20	45 - 400
	ppm wet wt	ppm dry wt

Toxic dose (methyl mercury)

1.76 mg Hg/kg body wt for 14 wks.
0.74 mg Hg/kg body wt for 40 wks.
0.46 mg Hg/kg body wt for 60 wks.
A cumulative dose of 20 mg methyl mercury/kg body wt is toxic.

Toxic Signs

Ataxia, inco-ordination, abnormal gait, muscle weakness, tremors and convulsions.

MERCURY

Horse: Tissue Levels

Diet	Liver	Kidney	Blood	Brain
Normal	< 0.1	< 0.1	< 0.01-0.10	< 0.01
Toxic (Chronic)	5.0-10.0 ppm	5.0-300 ppm	2.0 -6.0 ppm	> 3.0 ppm

Toxicity - Normal diet < 0.1 ppm
Toxic diet (chronic) 0.40 mg/kg body wt - methyl mercury

Toxic signs - reduced appetite, weight loss, renal disturbance and neurological dysfunction. Laminitis and bilaterally symmetrical exudative dermatitis.

MERCURY

Pigs: Tissue Levels

	Diet	Liver	Kidney	Brain
Normal	< 0.1	<0.01-0.03	<0.01-0.09	< 0.10
High		1.0 -6.8	1.0 -8.0	0.5-5.0
Toxic		5.0 -150	10.0-200	5.0-25
	ppm	ppm wet wt		

Toxicity signs - anorexia, loss of weight, central nervous system depression, weakness, abnormal gain and inco-ordination. Vomiting and diarrhea. Signs of toxicity may be delayed up to 3 weeks after a single toxic dose.

Toxic dose - Acute 20 mg/kg body wt methyl mercury
Chronic 0.2 mg/kg body wt methyl mercury

Mercury is cumulative , toxicity occurring after about 20 mg/kg body wt has been consumed.

Interactions - Selenium counteracts mercury toxicity, resulting in an increased accumulation of inorganic mercury in the liver and spleen.

MERCURY

Poultry: Tissue Levels

	Liver	Kidney	Brain	Blood
Normal	0.01-0.10	0.05-0.30	0.1	0.1
High	1.0 -10	2.0 -10	0.2-10	0.2-0.5
Toxic	3.0 - 130	5.0 -90	0.5-40	0.1-12

ppm wet wt

	Muscle	Feathers	Eggs White	Eggs Yolk	Eggs Total
Normal	0.008-0.10	0.07-0.10	0.03	0.03	0.03
High	1.0 -2.0	0.40-1.0	5 -10	0.5-3.0	0.1-0.9
Toxic	5.0 -14.0	6.0 -12.0	10-30	0.9-6.0	1.0-2.0

ppm wet wt

Tissue levels are a good indicator of exposure to mercury, however, in view of the extremely high levels found in some experimental birds which showed no signs of toxicity, a diagnosis of mercury toxicity should not be based on tissue residues alone.

Distribution of mercury in the body depends on the form in which it is ingested. Organic mercury ingestion leads to higher blood and brain levels with equal amounts in liver and kidney.

Inorganic mercury leads to high kidney concentrations. The proportion of Hg in egg yolk v. white and total egg Hg v. methyl Hg varies with the form of ingested mercury.

Toxicity -

Toxic dose (lethal)	Single IV injection	Dietary	Water
Inorganic Hg	2.6	50-100	500
Alkyl (methyl) Hg	30	5-80	
	mg Hg/kg body wt	ppm	

Dietary methyl mercury is more toxic than inorganic Hg. Young birds are more susceptible.

Mercury has a biological half life of 30 days.

Signs of Toxicity - Inorganic: anorexia, necrosis of gastro intestinal tract, nephrosis. Organic: abnormal neurological patterns, egg shell thinning.

MERCURY

Sheep: Tissue Levels

Diet	Liver	Kidney	Blood	Wool
Normal	< 0.1	< 0.1	< 0.1	< 0.1
High	7 - 30	18 - 200	0.2 - 2.0	0.2 - 10
Toxic	10 - 60	20 - 200		

ppm wet wt

Sheep appear to accumulate less mercury residues than cattle fed the same dietary level.

MOLYBDENUM

Cattle: Tissue Levels

	Liver	Kidney	Milk	Blood
Normal level (at normal Cu level)	0.57-1.14	0.22-0.57	0.018-0.120 (0.073 av)	0.01-0.05
Toxic (Cu deficient)	1.4 -100	1.15-2.60		0.10-0.47
		ppm wet wt		

Toxicity - levels increase with increased Mo intake but are not toxic unless Cu levels are low. (Non toxic levels of blood 10.0 ppm and liver 1000 ppm have been recorded).

Interactions - toxicity is dependent on copper:molybdenum:sulphur (protein) in diet. Low Cu and high sulphur aggravate toxicity.

Dietary levels -

Normal pasture		Mo 0.5- 3.5 ppm	Cu 8-11 ppm
Toxic condition	(cattle)	5.0- 6.0	8-11
	(sheep)	10.0-12.0	8-11

High levels generally occur only in peat or organic soils.
Mo pasture levels lowest in Winter rising from April to peak in September.
Liming soil increases plant Mo uptake - high levels often occurring in plants growing in alkaline sloughs.
Cu/Mo ratio diet: ideal 6:1, borderline 2:1-3:1, toxic < 2:1.
Dietary Mo above 10 ppm can cause toxicity regardless of Cu intake.

Prevention of Toxicity - 0.5 - 2.5 g $CuSO_4$ daily will protect against 150 ppm Mo.

Deficiency - no essential functions have been identified for molybdenum in ruminant nutrition.

MOLYBDENUM

Chickens: Tissue Levels

	Diet	Liver	Kidney	Egg (Yolk)
Deficient	< 0.02	0.3		
Marginal				
Adequate	0.03-1.0	0.4-0.8	1-2	0.03-0.08
High	3 -10	1 -4		0.30-0.70
Toxic	> 200	6.0-10		
		ppm wet wt		ppm wet wt

Deficiency - Mo is an essential trace element for poultry. Mo deficiency is thought to be partially responsible for a poor hatchability syndrome with weak chicks having clubbed down and long ginger hairs. It is involved in feather development, and is also thought to be partially responsible for the scabby hip syndrome and femoral degeneration. More recent work indicates Mo responsive conditions must be due to an interaction with at least one other unknown nutritional disorder or disease syndrome.

Mo is a component of xanthine oxidase and aldehyde dehydrogenase.

Interactions - availability is affected by Cu, Zn, Mn, Sulfate, Cd, W, F, Fe and methionine.

Deficiency treatment - In some cases 40 µg Mo (as ammonium molybdate) single oral dose, has alleviated mortality in club down chicks and improved feather growth (in males only). Addition of 0.2-2.5 ppm Mo to diet has prevented scabby hip syndrome.

0.2 to 0.5 µg Mo (as ammonium molybdate) intramuscularly corrected scabby hip syndrome and restored normal feathering in some experiments.

Toxicity - dietary levels of 3-10 ppm Mo have inconsistently impaired reproductive performance. Dietary levels greater than 200 ppm Mo reduce growth rate.

MOLYBDENUM

Pigs: Tissue Levels

	Diet	Liver
Sows	Normal	0.6-1.3 ppm
Slaughter Pigs	Normal	0.8-1.9 ppm

A toxic diet of molybdenum is 1000 ppm.

NICKEL

Cattle: Tissue Levels

	Diet	Liver	Kidney	Milk
Deficient	.002-.080		0.02	
Marginal	0.1			
Adequate	1.0 -10.0	0.2-0.6	0.15-0.50	0.02-0.10
High	100 -1000	0.2-0.6	6.8	
Toxic	> 1500			0.02-0.10
	ppm	ppm wet wt		

Normal Blood Serum - Nickel

Cat	1.5-6.4 ug/L	Goat	2.7-4.4 ug/L
Cattle	1.7-4.4	Horse	1.3-2.5
Dog	1.8-4.2	Pig	4.2-5.6
	Rabbit	6.5-14.0 ug/L	

Dietary levels - pastures contain 0.5-3.5 ppm Ni, grains contain 0.3-0.6 ppm Ni.

Deficiency - Nickel deficiency seems unlikely in farm animals, although little is known about the bioavailability of Ni.

Deficiency signs (<0.05 ppm Ni in diet)

Lambs - nickel deficient lambs have shown reduced liver copper stores and elevated liver iron levels. Nickel deficiency reduces the rumen bacterial urease activity and deficient lambs grow less rapidly.

Pigs - reduced weight gain, delayed sexual maturity, higher piglet mortality. Parakeratosis with lowered Zn levels in hair and liver.

Toxicity - high levels reduce palatability of feed and reduce intake - chloride more toxic than carbonate.

Interactions - apparent Ni-Zn interaction.

NICKEL

Poultry: Tissue Levels

	Diet	Liver	Kidney	Muscle	Bone
Deficient	< 0.05				
Marginal					
Adequate		0.10	0.13	0.14	0.10
High	100- 300	0.36	4.2	0.25-0.40	0.90-1.0
Toxic	700-1100	1.0-1.4	9.7-12.0	0.60-2.60	3.80-6.0
	ppm	ppm wet wt		ppm wet wt	

Toxicity - excess cobalt enhances Ni toxicity (additive).
No specific signs are noted with a toxicity other than reduced growth rate.
Reduced growth rate caused by excessive Ni is significantly reduced by feeding high protein levels.
Studies suggest nickel can be teratogenic in chickens.

Deficiency - Ni interacts with Cu, Zn and Fe.
Signs - decreased yellow pigmentation of shank skin, thickened legs, swollen hocks, dermatitis of shank skin and anaemia.

SELENIUM

Cat: Tissue Levels

	Diet	Liver	Kidney	Whole Blood
Deficient				
Marginal				
Adequate	0.3	0.26-0.54	0.77-1.14	1.7- 2.5
High	4.7	2.0 -4.6	4.2 -9.4	9.1-19.7
	ppm		ppm wet wt	ppm dry wt

The incidence of selenium deficiency in cats is not yet well defined.

SELENIUM

Cattle: Tissue Levels

	Diet	Liver	Kidney	Serum
Deficient	< 0.10	0.02-0.17	0.18-0.40	0.002-0.008
Marginal	0.10-0.18	0.12-0.25	0.40-1.00	0.020-0.040
Adequate	0.25-2.00	0.25-0.50	1.00-1.50	0.070-0.300
High	3.0 -4.0	0.75-1.25	2.00-2.50	2.5 -3.5
Toxic (chronic)	> 5.0-20	1.25-3.8	2.5 -5.0	3.5 -4.1
(acute)		7.0 -10.0	1.0 -3.0	
	ppm dry wt	ppm wet wt (dry wt x 3.5 approx.)		

	Hair	Milk	Muscle	Hooves
Deficient	0.06-0.23	0.004-0.005	0.010-0.050	
Marginal	0.23-0.50	0.011-0.018	0.050-0.070	
Adequate	0.50-1.32	0.030-0.050	0.070-0.150	
High	1.40-30.0	0.070-1.270	0.250-0.500	
Toxic	1.40-45.0	0.080-	0.50 -1.50	10.0
	ppm dry wt	ppm wet wt		ppm

Fetal liver contains double level of maternal liver on dry wt basis (fetal ppm wet wt x 6 = dry wt). We presently consider 0.30 ppm Se wet wt in the fetal liver to be an adequate level.
Elevated CPK (>100 IU/L) is a good indicator of sub-clinical WMD
Elevated LDH and SGOT levels occur in clinical cases.
Serum contains approximately 30% of the whole blood Se in cattle with an adequate Se intake. Adequate whole blood Se > 0.090 ppm.

Dietary requirement - milking cow 4.0-6.0 mg Se/day.

Deficiency signs - acute selenium deficiency can cause white muscle disease (WMD), diarrhea, muscle stiffness and occasionally recumbancy particularly in parturient cows (similar to milk fever syndrome). Sudden death due to cardiac failure with no prior signs of sickness.

Marginal selenium deficiency can result in retained placentas, abortions, reduced fertility, decreased growth rate, decreased immune response.

Selenium - Cattle contd.

Deficiency prevention - selenized salt or mineral mix containing 25-75 ppm Se free choice or 40 g/day/mature cow. Intraruminal pellets - two 30 g pellets containing 10% Se, last 18 months.

Treatment - injectable (e.g. Dystosel), one injection (0.13 mg Se/kg body wt) will maintain body reserves of 40-60 days. Treatment should be followed by a prevention program to prevent recurrence.

Blood Glutathione Peroxidase Activity (GSH-Px u moles/min at 37°c)

	Serum Se	GSH-Px
Deficient	0.009-0.050	0.2-10.0
Marginal	0.009-0.072	10-19
Adequate	0.104	19-36

Reports of a non-selenium dependent glutathione peroxidase have appeared in the literature. Recent findings indicate that bovine erythrocytes contain only the selenium dependent form thus substantiating blood glutathione peroxidase activity as a reliable indicator of selenium status. Liver and kidney however appear to contain large amounts of non-selenium glutathione peroxidase.

There appears to be a time lag of about 9 days before supplemented selenium maximises GSH-Px levels.

Toxicity - LD_{50} chronic 0.4-0.5 mg/kg body wt or 50 mg/day/cow
subacute 10-20 ppm for 7-8 weeks
acute injectable Se 7½-15 times therapeutic dose.

Hair is not a good indicator of toxicity as sulphur (protein) intake affects deposition in hair.

Signs of toxicity - "blind staggers" or "alkali disease", loss of hair, lameness, with cracked or deformed hooves.

Interactions - Arsenic, cadmium, copper, lead, mercury, silver, telurium, zinc and to a lesser extent sulphate reduce the toxicity of selenium. They can also induce a deficiency of selenium, thus it may be necessary to increase Se supplementation when As or Cu supplementation is being used or when animals are exposed to industrial pollution.

Selenium - Cattle contd.

Interactions

High linoleic acid intake (e.g. barley) increases incidence of WMD when Se intake marginal.

Treatment of high moisture corn with proprionic acid to retard spoilage reduces the vitamin E content with resultant increased WMD.

Plant uptake - Selenium in well-aerated, alkaline soils is more readily available for plant uptake than similar quantities in poorly aerated acid soils.

SELENIUM

Dogs: Tissue Levels

	Diet	Liver	Kidney	Blood
Deficient	0.01-0.20	0.10-0.30		
Marginal	0.20-0.50	0.30-0.50		0.22
Adequate	0.51-1.0	1.00-	1.00-1.50	
Borderline				
Toxic				
	ppm dry wt	ppm wet wt		

Deficiency is implicated in hip dysplacia and reduced immune response.

SELENIUM

Horse: Tissue Levels

	Diet	Liver	Kidney	Blood(serum)
Deficient	0.01	0.16	0.58	0.008-0.053
Marginal				0.053-0.120
Adequate	0.2-2.0	0.30-1.0	0.70-2.0	0.140-0.250
High				0.350-
Toxic	30	10.0		
	ppm	ppm wet wt		ppm

	Milk	Hair
Deficient	0.005-0.009	< 0.50
Marginal	0.008-0.015	
Adequate	0.015-0.040	1.0-3.0
High		5.0-7.0
Toxic		7.0-30.0
	ppm	ppm dry wt

Normal fetal liver Se seems to be in the region of 0.29-0.46 ppm wet wt. (A liver level of 0.12 ppm has been recorded in foal when mare fed selenized salt free choice (25 ppm) and injected with dystocel 4 weeks before foaling.)

Normal serum vitamin E - 4.2-8.7 µg/ml.

Blood level	SGOT	CPK	ɣGT	Erythrocyte GSH-Px
Se deficient	Elevated	Elevated	50-120	< 9.0
Se adequate	200-400	20-100	20-30	9.0 -
	IU/L	IU/L	IU/L	µ moles/ml

Deficiency - signs - muscular dystrophy in foals. Myocardial and skeletal muscle disease often with steatitis. Azoturoa (tying up syndrome). Reproductive disorders - pyometritis, repeat breedings, early embryonic death, abortions and sudden death of foals.

Prevention - 1 mg Se + 200 IU vitamin E/day. Daily Se requirement 2.4 µg/kg/day.

Selenium - Horse contd.

Toxicity

Toxic dose = 3.3 ug/kg/day

Chronic toxicity signs - lameness, cracking and sloughing of hooves. Loss of tail and mane hair, dullness, emaciation, depraved appetite.

SELENIUM

Pigs: Tissue Levels

	Diet	Liver	Kidney	Serum
Deficient	0.01-0.08	0.03-0.10	0.40-0.77	0.005-0.060
Marginal	0.10-0.20	0.12-0.25	0.77-1.10	0.060-0.100
Adequate	0.20-0.80	0.30-0.80	1.50-2.90	0.120-0.300
High	3.0 -4.0	3.0 -12.0	3.0 -18.0	
Toxic	7.5 -	12.0-120	18 -90	0.50 -0.90
	ppm dry wt (growing pigs lower end of range - sows higher end of range.)	ppm wet wt		

	Muscle	Milk	Hair
Deficient	0.020-0.050	0.013-0.043	0.18-0.22
Marginal	0.050-0.075		
Adequate	0.100-0.250	0.120-0.20	0.24
High	0.55		
Toxic	2.00		
	ppm wet wt	ppm at 7^{th} day	ppm dry wt

Piglets from normal sows are born with a reserve of Se in the liver (1.6-2.6 ppm wet wt) which depletes during the suckling period (to a minimum adequate level of 0.30 ppm) i.e. sows milk does not supply sufficient Se for the needs of the nursing pig. Serum levels may drop from 0.10 to a critical 0.04 ppm at weaning, but should return to normal when eating solid food (Se 0.2 ppm). This is a point in favor of early weaning.

Selenium is distributed evenly throughout the liver.

Glutathione Peroxidase Activity GSH-Px

Selenium	Erythrocytes	Plasma
Deficient	< 50	0.25-2.0
Marginal	50 - 100	2.0 -3.0
Adequate	100 - 200	3.0 -8.0
	μ moles GSH oxidised /min/g Hb	n moles NADPH oxidized/ min/mg protein

Deficiency - Stress and exercise seem to hasten development of signs.

Selenium - Pigs contd.

Signs of Deficiency - Hepatosis dietetica, mulberry heart disease or degeneration of skeletal muscles with resultant sudden death mainly in fast growing animals. Iron injections can cause iron toxicity in Se deficient piglets. Lowered disease resistance and possible reproduction problems have been ascribed to Se deficiency.

Minimum recommended diet 0.15 - 0.2 ppm Se + 10 IU vitamin E
or 0.20 - 2.0 ppm Se + 5 IU vitamin E

Treatment of marginal Se deficiency

One injection: 0.25 mg Se/pig before 7 days old
0.06 mg Se/kg body wt at 40 days old.

Minimum dietary level 0.2 ppm Se.

Treatment of severe deficiency

Injection: medium 0.22 mg Se + 15 IU vit. E/kg body wt.
high 1.10 mg Se + 75 IU vit. E/kg body wt.

Diet supplementation to 0.60 ppm Se + 30 IU vit. E/kg.

Se more readily absorbed from drinking water than from feed.
Elevated CPK ($>$300 IU/ml) is a good indicator of subclinical muscular myopathy.

Toxicity - toxic dose as Na_2SeO_3 17 µg/kg body wt (8 µg/lb)

Poor conception caused by 10-20 ppm Se in diet for 7-8 weeks. Animals showing toxic signs generally have higher liver than kidney levels.
Do not give high level injections to sows during last month of gestation as this may induce Se toxicity in the feti.

Interactions - Arsenic and sulphur reduce Se retention in tissues.
Silver reduces blood Se levels but increases liver retention.
Proprionic acid treatment of high moisture corn destroys vitamin E.
Se-vitamin E supplementation reduces incidence of swine dysentry in Se deficient pigs.
Manganese deficiency aggravates Se deficiency.
Riboflavin (vitamin B_2) is involved in the Glutathione Peroxidase system a deficiency of which may be implicated in WMD.

SELENIUM

Poultry: Tissue Levels

	Diet	Liver	Kidney	Blood
Deficient	0.01-0.06	0.05-0.25	0.10-0.40	0.02-0.85
Marginal	0.06-0.10	0.25-0.35	0.40-0.50	0.85-0.13
Adequate	0.20-1.0	0.35-1.00	0.50-0.70	0.13-0.20
High	3.0 -5.0	2.00-4.0	1.5 -2.5	
Toxic	5.0 -10.0	4.0 -		
	ppm dry wt	ppm wet wt (x 4 = dry wt approx.)		

	Muscle	Whole Eggs	Egg White	Egg Yolk
Deficient	0.020-0.065	-0.04	0.01-0.05	0.05-0.20
Marginal	0.065-0.075	0.06-0.10	-0.05	0.20-0.40
Adequate	0.100-0.22	0.25-1.00	0.05-0.25	0.40-1.00
High	0.400-0.50	1.50-2.00	0.32-0.80	1.00-2.80
Toxic		2.50	1.0 -1.4	3.3 -4.0
	ppm wet wt		(white:yolk = 2:1)	

Recommended minimum dietary level:

Chicks 0.1 ppm Se + 30 IU vit.E/kg or 0.2-2.0 ppm Se

Poults 0.2 ppm Se + 10 IU vit.E/kg or 0.3-2.0 ppm Se

Minimum adequate diet for chicks 0.1 ppm Se + 5 IU vit. E.

Average level vitamin E in poultry feeds 3-5 IU.

Deficiency signs: Exudative diathesis. Chronic Se deficiency results in reduced growth rate, egg production, hatchability and fertility and reduced immunity to infectious diseases and coccidiosis.

Toxic levels - Single dose 80 mg/kg

Acute oral LD_{50} 33 mg/kg body weight

Max tolerated dose 15 mg/kg body wt/day as selenite.

A diet of 5-10 ppm Se did not affect the laying hen but embryonic development was adversely affected.

80 ppm in feed terminates egg laying with subsequent 30% mortality in hens.

Selenium - Poultry contd.

Availability and interactions -
Se availability reduced by arsenicals, high protein, linseed oil, unsaturated fats, lead, cobalt, mercury, cadmium, tellurium, silver, copper and tin.
Dietary ascorbic acid (100 ppm) increases Se absorbtion from diet.

Utilization may be dependant on the antioxidant, Vitamin E or B-6 levels in the diet.

Availability of the Se in various dietary forms is not at all clear due to literature discrepancies. Ranges reported as a percentage of the availability of Se as socium selenite (taken as 100%) are as follows:

Substance	Se Availability
Fish meal	33 - 60%
Soybean meal	15 - 65%
Grains	20 - 85%
Selenomethionine	70 - 80%

In vegetable foods Se seems to be present mainly as selenomethionine.

SELENIUM

Rabbit: Tissue Levels

	Diet	Liver	Kidney	Blood	Muscle
Deficient	0.10				
Marginal		1.00		0.35	
Adequate	0.20	1.07	1.58	0.35	0.18
High					
Toxic		7.04	12.23	5.06	1.35
	ppm		ppm wet wt		

Toxicity - LD_{50}/24 hr of Sodium selenite 2.53 mg/kg body weight
Sodium with vit. E 2.73 mg/kg body weight

Selenium metabolism in the rabbit appears to differ from that of other species. Muscular dystrophy will develop when vitamin E deficient diets are fed, but selenium has not been found to partially protect against, or alleviate the condition as is the case in other species. This would indicate that protection against peroxide damage of tissues in the rabbit is more dependent on vitamin E than selenium. It seems more likely, however, that the higher tissue levels of selenium found in normal rabbits indicates a more efficient use and storage mechanism than in other species. The possibility then exists that the experimental rabbits used to evaluate selenium/vitamin E metabolism were not sufficiently depleted of selenium prior to commencing the investigation. Rabbits would appear to be less susceptible to selenium d eficiency than all other domestic species.

All GSH-Px circulating in the blood is of the Se dependent type - however the liver and kidney contain 43% and 40% respectively of non Se dependent GSH-Px, this and the total GSH-Px is more than in other species.

SELENIUM

Sheep: Tissue Levels

	Diet	Liver	Kidney	Serum
Deficient	0.02-0.10	0.005-0.100	0.046-0.600	0.006-0.030
Marginal	0.10-0.18	0.150-0.250	0.700-1.10	0.030-0.050
Adequate	0.20-0.50	0.250-1.0	0.90 -3.00	0.080-0.500
High	3.00-5.00	2.00 -10.0	4.0 - 6.0	
Toxic	5.00-25.0	15.0 -30.0	6.0 - 15.0	3.0
	ppm	ppm wet wt		

	Milk	Wool	Muscle
Deficient	0.002-0.020	0.03-0.30	0.010-0.025
Marginal		0.03-0.60	0.025-0.090
Adequate	0.025-0.040	0.70-4.00	0.090-0.400
High			0.400-0.600
Toxic			0.60 - 2000 ppm wet wt

Glutathione Peroxidase Activity (GSH-Px)

Selenium	Liver	Erythrocytes
Deficient	100 - 150	2.0 - 6.9
Marginal	150 - 240	8 - 30
Adequate	300 - 500	60 - 180

(Erythrocytes - u moles NaDPH reduced/g Hb/min at 37°c)
(Liver EU/g fresh tissue - On. J. An. Sci. 42 (4) p 984)

Erythrocytes GSH-Px seems to correlate best with Se status of sheep.

GSH-Px levels do not correlate with incidence of WMD.

Elevated CPK levels are best indicators of subclinical WMD.

Elevated LDH and SGOT levels indicate muscular damage.

Deficiency - selenium deficiency reduces reproductive performance and is partially responsible for white muscle disease (WMD).

Se deficiency reduces immune response - supplementation above nutritional requirements increases number of IgM producing cells and synthesis of IgM antibody. Se deficient sheep have lowered resistance to bacterial diseases and parasites.

Selenium - Sheep contd.

Selenium supplementation has been found to prevent peridontal disease affecting the molars of sheep in selenium deficient areas in New Zealand.

Therapeutic oral dose - 1 mg/kg/wk
5 mg/kg/month
Iron/Se bullet.

Deficiency prevention - free choice (or 12.6 g/day/ewe) selenized salt or mineral mix containing 26-75 ppm Se.
Injectable Se treat once every 3-4 months.

Drench or inject ewes and rams 3-4 weeks before breeding and 3-4 weeks prior to lambing. Add Se mineral mix to grain ration when fed at lambing time. Organic or naturally available Se is thought to be more available than inorganic Se.

Interactions - Se alone will not prevent the occurrence of WMD. Vitamin E must also be available. 10 ppm vitamin E required to prevent WMD at 0.1 ppm Se level in diet. Vitamin E in legumes appears to be less available than that in grass. May be amino acid-sulphur interaction affecting Se absorbtion from the gut.

Cyanide from plant cyanogenic glycosides can induce WMD in Se deficient lambs.

Super phosphate fertilization of pastures leads to reduced Se levels in grazing sheep but not in the pasture.

Cobalt deficiency increases susceptability to Se toxicity.

Toxicity - Toxic dose as Na_2SeO_3, LD_{50} Intramuscular 0.45 mg/kg
Parentally 5.0 mg/kg
Orally 10-15 mg/kg
Chronic toxic dose 0.08 mg Se/kg/day for 1 year.

TUNGSTEN

Poultry: Tissue Levels

	Diet	Liver	Blood
Deficient			
Adequate	0.2	0.33	0.1
High	1.0	3.5-15	
Toxic		24 -30	15.4
	ppm dry wt	ppm wet wt	ppm

Deficiency - the requirement to tungsten by animals is presently unknown.

Interactions - Tungsten inhibits the absorption of Mo from the digestive tract and may interfere with some enzyme syntheses particularly xanthine dehydrogenase.

Toxicity

Signs of toxicity - reduced feed intake and weight gain, diarrhea with death within one day of onset. Distress with labored breathing occurring only about 1 hour before death.

Emaciation, dehydration and extensive muscle hemorrhaging are apparent on post mortem examination together with petechial haemorrhages on the gizzard, proventriculus and in the brain, heart and kidney.

URANIUM

Ruminants

Estimated maximum safe dietary intake of natural uranium

	Cattle	Sheep
Feed	30 ppm dry wt	20 ppm dry wt
Water	10 ppm	20 ppm

Estimated daily intake of natural uranium by sheep and cattle to produce various effects:

Daily intake of U		Effect
Sheep	Cattle	
0.05g	0.4g	Slight malaise in sheep, transient depression of milk yields in cows
25g	200g	Meat unfit for human consumption
-	2 kg	Milk unfit for human consumption

Toxicity: Cattle

75 ppm U in water as uranyl nitrate resulted in general deterioration of health for 2 weeks then no further effect.

A minimal deleterious dose is ESTIMATED to be 1/10 this level = 0.4g U/ day/mature cow. Ref. Garner. Health Physics 1963 (9) 597-605.

Deficiency - no evidence to suggest uranium acts as an essential trace element in mammals.

Post Glacial uranium deposits generally occur in alkaline flats or sloughs and highly organic areas such as peat bogs. They are usually associated with high Mo and Se deposits.

Normal bovine liver uranium = 0.0008 ppm dry wt.

URANIUM

Dogs: Tissue Levels

	Diet	Kidney	Bone
High	0.10-0.20	0.2-1.0	
Toxic	2.1 -	1.0-2.3	
	mg/kg body wt/day	ppm wet wt	

Toxic effects of uranium - chronic

Changes in blood cell morphology.

Disturbance of thyroid function.

Increased basal metabolism, changes in hepatic function.

Hematopoietic deficiency and renal damage.

Most uranium is deposited in the bone (300 day ½ life) or kidneys (15 day ½ life).

Albumin appears in urine as kidney damage commences.

References

Voegtlin, C. and Hodge, H.C.: Pharmacology and Toxicology of Uranium Compounds, C. Voegtlin, H.C. Hodge, Editors, McGraw-Hill Company, New York. National Nuclear Energy Series, 1949.

Yuile, C.L.: Animal Experiments in Uranium, Plutonium, Transplutonic Elements, H.C. Hodge, J.W. Stannard, J.B. Hursh, Editors, Springer-Verlag, New York. Handbook of Experimental Pharmacology 36, 165-196, 1973.

Durban, P.W.: Metabolism and Effects of Uranium in Animals, in Health Experience with Uranium, ERDA Publication 93, US Energy Research and Development, pg 76, 1975.

VANADIUM

Chicken: Tissue Levels

	Diet	Liver	Kidney	Bone (Tibia)	Eggs (Yolk)
Deficient	0.010-0.035				
Marginal					
Adequate	0.10 -3.0	0.018-0.038	0.180	1.3-6.3	0.002-0.003
High	10 -30			5.8-8.6	
Toxic	100 -800			10 -15	
	ppm dry wt	ppm wet wt		ppm dry wt	ppm wet wt

Ducks: Tissue Levels

	Diet	Liver	Kidney	Bone	Eggs
Normal	1-10	0.013-0.080	0.0007-0.002	0.080-0.23	<0.0002
High	100	0.540-0.760	0.230 -0.320	0.230-5.50	0.059-0.068
			ppm wet wt		

Toxicity - Increased protein in the diet protects slightly against vanadium toxicity. Increased lactose enhances toxicity. Toxicity is decreased by Cr, cottonseed meal, dehydrated grass and ascorbic acid.

Deficiency - Vanadium is an essential trace element for birds. Deficiency reduces wing and tail feather growth and body growth rate. Vanadium is involved in body lipid metabolism.

Blood and bone iron levels tend to increase in V deficient chicks. Bone development is retarded in deficient chicks.

Naturally occurring deficiencies have not been reported but feedstuffs frequently contain less than the estimated minimum requirement of 0.1 ppm V.

VANADIUM

Sheep: Tissue Levels

	Diet	Liver	Kidney
Deficient			
Marginal			
Adequate	0.1-10.0	0.10-0.22	0.20-0.47
High	100-200	0.84-3.0	3.50-11.6
Toxic	400-800		
		ppm dry wt	

Cattle: Tissue Levels

		Liver	Kidney	Milk
Deficient				
Normal		0.006-0.007		0.1-0.2
Toxic	10-20 mg/kg day	0.3 -5.1	4.2-40.0	0.1-0.2
		ppm wet wt		ppb

Deficiency - Vanadium deficiency reduces growth rate and reproductive performance. V is an essential trace element for animals.
V is poorly absorbed from soils by most plants - spinach, parsley and mushroom absorb higher amounts.

Soil average 100-200 ppm
Cereal grains 0.0007-0.014 ppm
Pastures 0.03-0.07 ppm

Toxicity - signs - diarrhea, refusal to eat, dehydration, emaciation, dry hair coat, inability or reluctance to rise and move.
Animals may be able to build up a resistance.
Effects are more pronounced in nutritionally unbalanced diets.
V occurs in certain phosphate supplements (100 ppm) and in fossil fuels.
Excess V accumulates in the liver but is excreted fairly rapidly upon reducing intake.

ZINC

Cattle: Tissue Levels

	Diet	Liver	Kidney	Serum
Deficient	2 -10	<20	16 -20	0.2-0.4
Low borderline	10 -30			0.5-0.6
Normal (adequate)	50 -100	25-50	18- 0	0.7-1.4
High borderline	1000-5000			
Toxic	>5000	>500	130-180	5.2-7.5
	ppm	ppm wet wt		ppm

	Hair	Milk	Feces	Bone (rib)
Deficient	80 -100		12.0-18.0	32-60
Low borderline				
Normal (adequate)	100-150	2.3-5.1	160 -220	70-250
High borderline				
Toxic		8.4	>8000	
	ppm dry wt	mg/l	ppm dry wt	ppm dry wt

All blood or serum levels reported in the literature prior to 1977 should be viewed with a good deal of suspicion due to sample contamination from 'rubber' stoppers.

Tissue levels are not a good guide to zinc deficiency in the bovine.

Infectious diseases seem to lower liver and serum levels but increase kidney levels. Bone levels decrease with increasing age of animal.

Muscle zinc: normal levels

Light muscle	30 ppm wet wt	69 ppm dry wt, fat free
Dark muscle	70 ppm wet wt	247 ppm dry wt, fat free

Average zinc level in B.C. forage

Legumes	23 ppm dry wt	Corn silage	24 ppm dry wt
Grass hay	21 ppm dry wt	Oat forage	22 ppm dry wt
		Grain	33 ppm dry wt

Zinc deficiency - dietary zinc requirement for dairy cattle = 45 ppm with 0.3% Ca. For each additional 0.1% Ca in diet add 16 ppm Zn.

Zinc - Cattle contd.

Deficiency signs - weak hoof horn with increased susceptability to interdigital dermatitis, foot rot, reduced conception rate, (The effect of deficiency is more severe in the male than female fertility. Spermatozoan maturation is severely affected.). Reduced growth rate and feed intake. Parakeratosis.

Hereditary zinc deficiency can occur in cattle.

Deficiency prevention - free choice or 65 g/day of a salt mineral mix containing 0.54% Zn.

Interactions - high dietary Cadmium (350 ppm) reduces zinc absorption in calves.
Zinc, copper and iron are mildly antagonistic.
High zinc levels reduce calcium metabolism.

Toxicity - toxicity of zinc in cattle is uncommon.
2% Zn in dairy feed have killed mature cows.
6 to 8 ppm Zn in water is thought to have adverse effects on cattle.
Young calves are more susceptible to poisoning than adult cattle.

ZINC

Dogs: Tissue Levels

	Diet	Liver	Kidney	Plasma
Deficient				0.20
Normal	50-100 ppm	30-50	16-30 ppm wet wt	0.60-1.00

Serum levels markedly increased by stress.
Serum levels decreased by hepatic disorders, hysterectomies, hypothyroidism and infections.
Females have higher serum zinc levels than males.
Alkaline phosphatase levels increased with increased zinc levels.
Decreased serum Zn was associated with reduced T. Protein.

Deficiency & Toxicity

Frequency and incidence in dogs is unknown.

ZINC

Horses: Tissue Levels

	Diet	Blood (whole)	Liver	Kidney
Deficient				
Lower borderline				
Normal	40-100	2.0-5.0	40-80	20-27
High borderline				
Toxic	3600	6.0-15.0	1300-1900	295-580
	ppm		ppm wet wt	

	Bone (rib)	Milk
Deficient		
Low borderline		
Normal	65- 75	4.0-2.0
High borderline		
Toxic	140-340	
	ppm dry wt fat free	ppm

Toxicity - in lab controlled experiments:

Foal threshold limit 60 mg/kg body wt/day

illness at 90 mg/kg body wt/day = 3600 ppm diet.

Tolerance level under field conditions with unknown interactions is definitely less.

Toxic signs - swelling at the epiphyseal region of the long bones, stiffness, lameness, anemia.

Interactions - kidney cortex levels increased with increasing Cd in diet.

ZINC

Pigs: Tissue Levels

	Diet	Liver	Kidney	Serum
Deficient	10-24	20-25		
Low borderline	30-50	25-35		0.4-0.6
Normal (adequate)	75-500	45-90	10-31	0.8-1.8
High borderline	1000-5000			
Toxic	> 5000			
	ppm	ppm wet wt		ppm

	Hair	Bone	Milk	Colostrum-23 days
Low		60-90	1.0-6.0	22.7-13.9
Normal	160-230	95-146		
High				
	ppm dry wt		ppm	ppm

Young growing pigs have zinc liver levels at the lower end of the range shown.

Zinc deficiency signs - parakeratosis. Reduced conception rate.

Toxicity - zinc toxicity is dependent on the form in which the zinc is available. e.g. > 0.1% zinc as lactate or carbonate is toxic.
> 0.5% zinc as oxide is toxic.
Young animals are more susceptible to toxicity than older animals.

Interactions -
Enteric infection decreases zinc retention in pigs - lowering liver and serum levels.
Increased copper supplementation increases Zn requirement and zinc storage in the liver. Increased zinc supplementation decreases Cu storage in the liver.
High calcium, soy-protein and phytate increase zinc deficiency.
High cadmium has no effect on zinc deficiency.
Ni and Co deficiency aggravate Zn deficiency.

ZINC

Poultry - Tissue Levels

	Diet	Liver	Kidney	Serum
Deficient	17	20-40		
Low borderline	50-80	20-40		
Normal (adequate)	100-200	40-70	22-32	2.1-2.7
High borderline	800-2000	(chicks-turkeys-quail)		
Toxic	2230-5000	200-700	300-800	
	ppm	ppm wet wt		ppm

	Feather			
	Feathers	Bone	Pancreas	Egg Yolk
Deficient		50		13-20 ppm
Low borderline				
Normal (adequate)	53-100	110-400	50-125	30-48 ppm
High borderline			1000-3500	0.7-1.0 mg/yolk
Toxic			1000-3500	
	ppm dry wt		ppm wet wt	

Tissue levels are not a good indicator of deficiency.

Diet - 100 ppm for 1st 3 weeks protects against feather defects in turkeys.
50 ppm is generally adequate after 3 weeks of age in turkeys.
Chickens require only half these amounts.
Laying hens: 10 ppm will maintain normal zinc content of body tissues.
70 ppm is required to maintain normal zinc content of eggs and newly hatched chicks.

Toxicity - is affected by the composition of the diet:
800 ppm Zn can be toxic in a sucrose-fish ration.
2000 ppm Zn is not toxic in a corn-fish ration.
Zinc carbonate is more toxic than zinc oxide.
20,000 ppm for 10 days induces moulting.
Dietary levels of zinc above 9000 ppm are unpalatable.

Interactions - high dietary calcium in the presence of phytic acid (soybean meal) reduces zinc absorption. Zinc is antagonistic to Cu, Fe, Mg and Mn.

ZINC

Rabbit: Tissue Levels

	Diet	Liver	Kidney	Blood (whole)
Deficient	0.2	55	20	32
Adequate	70	30-80	10-30	32
Toxic				
	ppm	ppm wet wt		ppm

Liver levels higher in newborn.

Tissue levels appear to be of little use for deficiency diagnosis.

Signs of deficiency - achromotrichia, dermatosis, hair loss, loss of hair pigment. Reduced fertility, loss of apetite.

ZINC

Sheep: Tissue Levels

	Diet	Liver	Kidney	Serum	Wool
Deficient	1-20			0.22-0.45	<70
Low borderline	30-50			0.40-0.80	
Normal (adequate)	50-100	30-75	12-30	0.80-1.50	70-130
High borderline	800-1000	400	1000	4.0 -5.0	
Toxic	2000			30.0-50.0	
	ppm	ppm wet wt		ppm	ppm

Plasma or serum zinc levels are affected by infection, trauma, low protein intake and pregnancy.

Toxic diet - acute 180 mg Zn/kg body wt.
chronic 20 mg Zn/kg body wt.

Zinc administered by drenching gun (50 mg/kg body wt) is more likely to cause toxicity than that administered in feed or intraruminal intubation.

Toxicity signs - diarrhea, loss of weight. High levels of dietary zinc cause pancreas damage.

Deficient diet - less than 0.05 mg Zn/kg body wt.

Deficiency signs - weak hoof horn with resultant increased susceptability to foot rot. Deficiency may adversely affect fertility (low conception, poor implantationand early embryonic death).
Skin lesions, frothy saliva, reduced food consumption and weight loss.
Reduced alkaline phosphatase levels in serum
deficient 1.0 - 3.0 sigma U/ml
adequate 4.0 - 5.0 sigma U/ml

Interactions - high Zn levels may protect against Cu toxicity.

Puls, R.
Veterinary trace mineral deficiency and toxicity information